Sri Sujanthy Rajaram, M.D, M.P.H
Editor

Critical Care Procedure Book

Nova Biomedical

Emergency and Intensive Care Medicine

NOVA

EMERGENCY AND INTENSIVE CARE MEDICINE

CRITICAL CARE PROCEDURE BOOK

No part of this digital document may be reproduced, stored in a retrieval system or transmitted in any form or by any means. The publisher has taken reasonable care in the preparation of this digital document, but makes no expressed or implied warranty of any kind and assumes no responsibility for any errors or omissions. No liability is assumed for incidental or consequential damages in connection with or arising out of information contained herein. This digital document is sold with the clear understanding that the publisher is not engaged in rendering legal, medical or any other professional services.

EMERGENCY AND INTENSIVE CARE MEDICINE

Additional books in this series can be found on Nova's website
under the Series tab.

Additional e-books in this series can be found on Nova's website
under the e-book tab.

EMERGENCY AND INTENSIVE CARE MEDICINE

CRITICAL CARE PROCEDURE BOOK

SRI SUJANTHY RAJARAM, MD, MPH
EDITOR

New York

Copyright © 2015 by Nova Science Publishers, Inc.

All rights reserved. No part of this book may be reproduced, stored in a retrieval system or transmitted in any form or by any means: electronic, electrostatic, magnetic, tape, mechanical photocopying, recording or otherwise without the written permission of the Publisher.

We have partnered with Copyright Clearance Center to make it easy for you to obtain permissions to reuse content from this publication. Simply navigate to this publication's page on Nova's website and locate the "Get Permission" button below the title description. This button is linked directly to the title's permission page on copyright.com. Alternatively, you can visit copyright.com and search by title, ISBN, or ISSN.

For further questions about using the service on copyright.com, please contact:
Copyright Clearance Center
Phone: +1-(978) 750-8400 Fax: +1-(978) 750-4470 E-mail: info@copyright.com.

NOTICE TO THE READER

The Publisher has taken reasonable care in the preparation of this book, but makes no expressed or implied warranty of any kind and assumes no responsibility for any errors or omissions. No liability is assumed for incidental or consequential damages in connection with or arising out of information contained in this book. The Publisher shall not be liable for any special, consequential, or exemplary damages resulting, in whole or in part, from the readers' use of, or reliance upon, this material. Any parts of this book based on government reports are so indicated and copyright is claimed for those parts to the extent applicable to compilations of such works.

Independent verification should be sought for any data, advice or recommendations contained in this book. In addition, no responsibility is assumed by the publisher for any injury and/or damage to persons or property arising from any methods, products, instructions, ideas or otherwise contained in this publication.

This publication is designed to provide accurate and authoritative information with regard to the subject matter covered herein. It is sold with the clear understanding that the Publisher is not engaged in rendering legal or any other professional services. If legal or any other expert assistance is required, the services of a competent person should be sought. FROM A DECLARATION OF PARTICIPANTS JOINTLY ADOPTED BY A COMMITTEE OF THE AMERICAN BAR ASSOCIATION AND A COMMITTEE OF PUBLISHERS.

Additional color graphics may be available in the e-book version of this book.

Library of Congress Cataloging-in-Publication Data

ISBN: 978-1-63482-418-7 (e-Book)
Library of Congress Control Number: 2015934137

Published by Nova Science Publishers, Inc. † New York

I dedicate the book to my family, my loving children and particularly my late mother who shaped my life and believed in me.

My loving mother,
Mrs. Sri Bala Saras Veluppillai
(3/13/1937 - 4/13/2015)

Contents

Preface		**xi**
Acknowledgments		**xiii**
Chapter 1	Arterial Cannulation *Jonathan D. Trager, DO,* *and Sri Sujanthy Rajaram, MD*	**1**
Chapter 2	Brain Tissue Oxygen Monitoring *M. Kamran Athar, MD* *and Jawad F. Kirmani, MD*	**7**
Chapter 3	Bronchoscopy *Ganga Ranasuriya, MD, Rohan Arya, MD,* *Adrian Pristas, MD and Archana Mishra, MD*	**11**
Chapter 4	Central Venous Catheter Placement *Ganga Ranasuriya, MD, Carol H. Choe, MD,* *Alaaeldin Soliman, MD and Sri Sujanthy Rajaram, MD*	**15**
Chapter 5	Cricothyroidotomy *Emily Damuth, MD and Sri Sujanthy Rajaram, MD*	**23**
Chapter 6	Chest Tube (Tube Thoracostomy) Placement *Rohan Arya, MD and Sri Sujanthy Rajaram ,MD*	**27**
Chapter 7	Esophageal Balloon Tamponade (Sengstaken-Blakemore tube placement) *Pedro Dammert, MD, Rohan Arya, MD,* *and Sri Sujanthy Rajaram, MD*	**33**
Chapter 8	ECMO for the Intensivist *Orlando Debesa, MD, Rohan Arya MD,* *and Sri Sujanthy Rajaram, MD*	**39**
Chapter 9	Feeding Tubes and Percutaneous Tubes *Shipali Pulimamidi, MD, Carol Choe, MD* *and Sri Sujanthy Rajaram, MD*	**45**

Chapter 10	FoCUS Echo in the Critical Care Setting *Ben Goodgame, MD, David Schrift, MD, Patricia E. Walley, Vivek Punjabi, MD and Sri Sujanthy Rajaram, MD*	**51**
Chapter 11	Intraosseous Access *Jonathan D. Trager, DO*	**63**
Chapter 12	Intra-Abdominal Pressure Monitoring *Sachin Mohan MD and Sri Sujanthy Rajaram MD*	**69**
Chapter 13	Intra-Aortic Balloon Pumping (IABP) *Jason Bartock MD, Carol Choe, MD and Sri Sujanthy Rajaram MD*	**73**
Chapter 14	Intracranial Pressure Monitoring *Michelle Ghobrial, MD, Jacqueline S. Urtecho, MD and Jawad F. Kirmani, MD*	**81**
Chapter 15	Intubation and Airway Monitoring *Munira Mehta, M.D, Mahesh Bhagat, MD, Yong-Bum Song, DPharm and Sri Sujanthy Rajaram, MD*	**89**
Chapter16	Jugular Venous Oxygen Saturation *Lauren Ng, MD, M. Kamran Athar, MD and Mohammad Moussavi, MD*	**99**
Chapter 17	Lumbar Puncture *Jessica Mitchell, MD, Fiorella Nawar, MD and Sri Sujanthy Rajaram MD*	**105**
Chapter 18	Noninvasive Positive Pressure Ventilation *Renato Blanco, MD Sri Sujanthy Rajaram, MD and Archana Mishra, MD*	**111**
Chapter 19	Paracentesis *Sachin Mohan, MD, Carol H. Choe, MD and Sri Sujanthy Rajaram, MD*	**115**
Chapter 20	Percutaneous Tracheostomy *Edward Peter Mossop, MD and Adel Bassily-Marcus, MD*	**121**
Chapter 21	Pulmonary Artery (Swan Ganz) Catheters *Nayan Desai, MD, Fiorella Nawar, MD, Mithil Gajera, MD, Munira Mehta, MD and Sri Sujanthy Rajaram, MD*	**131**
Chapter 22	Renal Replacement Therapy in the ICU *Rohan Arya, MD, Rajaram Kandasamy, MD and Sri Sujanthy Rajaram, MD*	**139**
Chapter 23	Targeted Temperature Management *Carol H. Choe, MD and Sri Sujanthy Rajaram, MD*	**143**

Chapter 24 Temporary Pacemakers **149**
Adarsh Srivastava, MD, Carol Choe, MD, and Sri Sujanthy Rajaram, MD

Chapter 25 Thoracentesis **153**
Rohan Arya, MD, Sri Sujanthy Rajaram, MD and Archana Mishra, MD

Chapter 26 Transcranial Doppler Monitoring **159**
M. Kamran Athar, MD

Chapter 27 Ultrasound in the Intensive Care Unit (ICU) **163**
David Shrift, MD and Sri Sujanthy Rajaram, MD

Chapter 28 Venous Oxygen Saturation Monitoring **167**
Philip Willsie, MD and Sri Sujanthy Rajaram, MD

Index **173**

Preface

Critical Care Medicine is a fascinating and unique subspecialty. Critical Care specialists require expertise in a broad range of procedures and deep understanding in all areas of medicine as well as surgery. As an Intensive Care Specialist we perform many procedures on critically ill patients. While training residents and fellows over the past years, I realized that there was no standard text book or guide book available for teaching and performing these lifesaving procedures at the bed side. This book is written to help the physician or practitioner at the bed side as a quick reference and guide. The book also helped many former residents and fellows to learn, get involved in publishing and educating colleagues whether in private practice or in academic medicine in their carriers. As Critical Care Physicians, any procedures done in our patients we must consider the risks involved and perform only when it is beneficial to the patient outcome. As healers we must consider the human values and respect the patient's autonomy.

I could not include some specialized chapters and procedures which are routinely performed by Intensivists such as mechanical ventilation and weaning methods because they are beyond the scope of this book. Critical Care Medicine is an evolving field that is branching out to several specialized areas of certification. I am hoping to include many other chapters in the subsequent editions of this book in the future.

I take this opportunity to thank all my teachers, fellows, residents, medical students and nurses who gave me the opportunity to teach and grow. Over the years I have learned much through teaching and training them.

As a working mother and physician, especially as an Intensivist with the demanding schedule and commitment, I spent enormous time in research and writing in an academic carrier. I would like to thank my husband, parents, and family for the unconditional support and understanding. I would like to dedicate this book to my three loving children Sanjev, Sankavi and Sweda who made my life complete as a successful mother and carrier woman.

Sri Sujanthy Rajaram, MD, MPH
Associate Professor of Medicine
Critical Care Intensivist & Sleep Specialist
JFK Medical Center, Edison, NJ, US and
Hackensack University Hospital, Hackensaack, NJ, US

Acknowledgments

Critical Care Procedure Book was written over three years.

Many of my former fellows who were in Critical Care Fellowship program at Cooper University Hospital co-authored several chapters. I would like to thank all the residents, fellows and my colleagues who co-authored the chapters.

Four of my former fellows had significant roles in correcting the contents throughout the years and helped in the production of the text book. I am very proud of them and glad that I got a chance to train these talented physicians not only in Critical Care but also in research. Special thanks go to Alisha Crawford who converted all hand drawn illustrations into publishable pictures.

1. **Ben Goodgame, MD**
   Emergency Medicine Consultant
   Lakes District Health Board, Rotorua Hospital, Emergency Department
   Private Bag 3023, Rotorua 3046, New Zealand
   Dr. Goodgame revised the contents format, modified the larger first version of the chapters into simple format and authored Focused ECHO chapter for an Intensivist at bed side.

2. **Rohan Arya, MD**
   Pulmonary Critical Care Fellow, Cooper University Hospital, Camden, New Jersey
   Dr. Arya hand drew most of our illustrations and figures, helped in the online submission of the book contents, modified some chapters' contents and co-authored many chapters.

3. **Carol Choe, MD**
   Critical Care Fellow, Cooper University Hospital, Camden, New Jersey
   Dr. Choe checked the contents for copyright, coordinated the online submission and authored many chapters in the book.

4. **Mithil Gajera, MD**
   Intensivist, Christiana Care Hospital, Wilmington, Delaware
   Dr. Gajera helped in checking the contents, references and authored a chapter.

5. **Alisha Crawford**
   Instructional Designer, Library Learning Commons, Cooper Medical School of Rowan University, Camden, New Jersey
   Alisha Crawford edited the hand drawn pictures and Figures into original publishable version.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 1

Arterial Cannulation

Jonathan D. Trager, DO[1],
and Sri Sujanthy Rajaram, MD, MPH[2*]
[1]St. Luke's University Health Network, Bethlehem, PA
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Arterial line placement is a common procedure for management of critically ill patients in various settings. Intra-arterial blood pressure measurement is more accurate than blood pressures obtained by non-invasive means, especially in the critically ill. Intra-arterial blood pressure management allows for the rapid recognition of changes in blood pressure, which is vital for patients on continuous infusions of vasoactive drugs. Arterial cannulation also allows for repeated arterial blood gas samples to be drawn without injury or discomfort to the patient. In unstable patients manual and automated cuff blood pressure measurements are often unreliable and an arterial line is recommended for continuos monitoring of blood pressure and titration of medications. Arterial lines can be placed at radial, axillary, femoral and dosalis pedis arteries. As you move more distally away from the heart, systolic blood pressure measurements tends to be higher and diastolic blood pressure drops, but the mean arterial blood pressure remains the same. Hence titrate the vasopressors for mean arterial blood pressure.

Indications

- Continuous direct blood pressure monitoring in unstable patients
- Frequent arterial blood gas sampling

* Email: sujanty@gmail.com.

- Unreliable or inaccurate indirect blood pressure monitoring
- Titration of vasoactive drugs and antihypertensive agents

Contraindications

Brachial and popliteteal artery cannulations are contraindicated because they are end arteries that brings blood supply to the upper and lower limbs respectively and any occlusion may result in limb ischemia.

Absolute contraindications

- Absent pulse
- Buerger disease (thromboangiitisobliterans)
- Full-thickness burns over the proposed cannulation site
- Inadequate circulation to the extremity
- Raynaud syndrome

Relative contraindications

- Anticoagulation or coagulopathy
- Atherosclerosis
- Inadequate collateral flow
- Infection at proposed cannulation site
- Partial-thickness burn at proposed cannulation site
- Previous surgery in area
- Synthetic vascular graft

Preparation

Necessary equipment includes the following:

- Sterile gloves
- Sterile gauze 4x4
- Chlorhexidine skin prep
- 1% Lidocaine without epinephrine in a 3-5mL syringe with a 25-27 gauge needle
- Arm board for brachial, radial, or ulnar cannulations
- Non-absorbable suture, 3-0 or 4-0
- Adhesive tape
- Sterile non-absorbable dressing
- Appropriate-sized cannula for the proposed artery
  - Radial artery cannula (Figure 1)
    - 20-gauge, 1¾-inch polyurethane catheter over 22-gauge introducer needle for catheter-over-needle technique

- 20-gauge peripheral artery catheter kit with integrated wire and catheter for modified Seldinger technique
- Femoral artery and Axillary artery cannula
  - Commercially-available kit (eg: Cook)
  - 18-gauge, 3-inch introducer needle or 20 or 22 gauge introducer for axillary artery
  - 4 French single-lumen catheter, 15 cm or longer
  - Guidewire, appropriately sized for catheter
- 3-way stopcock
- Pressure transducer kit
- Pressure tubing
- 500- to 1000-mL bag of normal saline

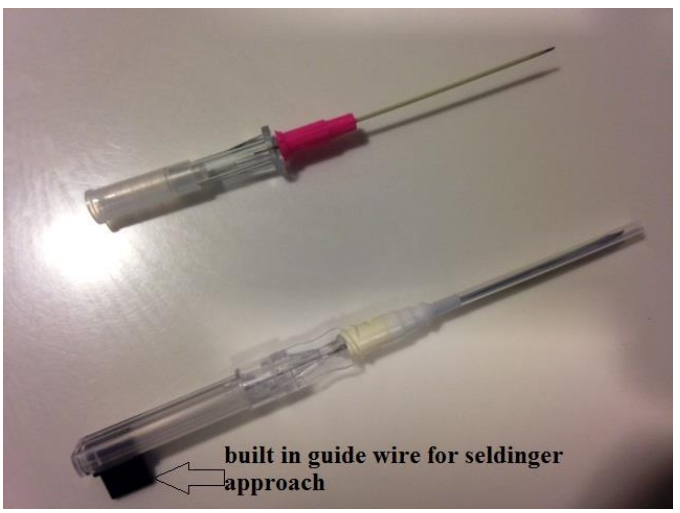


Figure 1. Two radial artery catheters.A 20-gauge catheter for the catheter-over-needle technique *(top)* and a 20-gauge catheter with guidewire for a modified Seldinger technique *(bottom).*

Procedure

The Allen Test

This test is performed to ensure that collateral circulation to the hand will be adequate if one of the arteries is cannulated.

1) The examiner occludes the radial artery using digital pressure, and the patient is asked to make a tight fist.
2) The hand is then opened, and the examiner assesses the hand for evidence of adequate blood flow.
3) The procedure is then repeated on the ulnar artery.

4) An abnormal (positive) Allen test is marked by continued presence of pallor 5-15 seconds after release of the artery. It is suggestive of inadequate collateral flow to the hand.
5) If return of color takes longer than 5-10 seconds, radial artery puncture should not be performed.

Radial Artery Cannulation

Once adequate collateral flow has been ascertained, arterial puncture may be performed.

1) Isolate the arterial pulsation on the palmar surface of the distal forearm. The radial artery is more superficial closer to the wrist and provides a more consistent cannulation.
2) Dorsiflexion of the wrist to approximately 60° over a towel or sandbag, preferably fixing the wrist to an arm board, will also significantly help isolate the artery.
3) Doppler or ultrasound use may facilitate percutaneous radial artery cannulations and minimize the number of punctures needed for placement.
4) Either via palpation or under direct visualization using ultrasound, direct the catheter over the radial artery in a 15-20° angle.
5) Make slight adjustments in angle and/or direction as needed in order to cannulate the artery.
6) Once blood return is noted, advance the catheter in the artery.
7) Connect the pressure tubing to the end of the catheter and secure the catheter in place.

Axillary and Femoral Artery Cannulation

The femoral artery and axillary artery are the commonly used vessels for prolonged arterial cannulation. Axillary artery closely resembles aortic pressure waveforms than those from any other peripheral site. Axillary arterial lines have the most accurate blood pressure monitoring in unstable patients.

1) Palpate the common femoral artery at the medial aspect of the thigh, just inferior to the inguinal ligament. The common femoral artery is located approximately one-third of the distance from the pubic symphysis to the anterior superior iliac spine (ASIS).
2) For axillary artery cannulation, palpate the axillary artery in the apex of the axilla or against the humerus. Position the arm at 90 degrees and abducted to open up the axilla.
3) Ultrasound guidance will confirm anatomy and improve likelihood of success.
4) Arterial puncture must always occur distal to the inguinal ligament to prevent uncontrolled hemorrhage into the pelvis or peritoneum, and for compressibility.
5) When puncturing the vessel, care must be taken to avoid the femoral nerve and vein, which create the lateral and medial borders, respectively.

6) Only the Seldinger technique is recommended for thiese sites, enabling placement of a 15- to 20-cm plastic catheter for prolonged monitoring. After the needle puncture pulsatile flow will confirm arterial puncture. Insert the guide wire through the hollow needle. Remove the needle and place the catheter through the guide wire. For arterial line placement incision and dilatation of the site may cause on going bleeding and not generally recommended. Skin and subcutaneous tissue may be dilated only if difficulty encountered during catheter placement.

Dorsalis Pedis Cannulation

The dorsalispedis artery continues from the anterior tibial artery. On the dorsum of the foot, the dorsalispedis artery lies in the subcutaneous tissue parallel to the extensor hallucislongus (EHL) tendon and between the EHL and the extensor digitorumlongus.

1) The artery should be cannulated in the superficial midfoot region.
2) Monitoring problems exist with cannulation of this artery. Pressures obtained with an electronic transducer attached to the dorsalispedis artery will be 5-20 mmHg higher than that of the radial artery andwill be delayed by 0.1-0.2 seconds

Local Puncture Site and Catheter Care

1) Once the catheter has been placed successfully, it should be advanced until the hub is in contact with the skin.
2) Connect the pressure tubing luer lock to the end of the catheter
3) Ensure all tubing connections are tight and secure
4) Secure the catheter by fastening it to the skin with suture material. 2-0 Silk and 4-0 nylon sutures provide the best anchoring.
5) After tying the catheter in place, a self-adhesive dressing is applied over the area.

Complications

Common complications:

- Temporary arterial occlusion (radial artery)
- Hematoma/bleeding

Rare complications:

- Infection – Due to pulsatile blood flow arterial line infection is very rare
- Thrombosis
- Ischemia
- Arteriovenous fistula
- Pseudoaneurysm

- Compartment syndrome
- Air embolism

References

Brzezinski M, Luisetti T, London MJ. Radial artery cannulation: a comprehensive review of recent anatomic and physiologic investigations. *AnesthAnalg*. Dec 2009;109(6):1763-81.

http://emcrit.org/pressure-set-up/

http://emedicine.medscape.com/article/1999586-overview

Milzman D, Janchar T. Arterial puncture and cannulation. In: Roberts JR, Hedges JR. *Clinical Procedures in Emergency Medicine*. 5th. Philidelphia: W.B. Saunders; 2010:349-363.

Scheer B, Perel A, Pfeiffer UJ. Clinical review: complications and risk factors of peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. *Crit Care*. Jun 2002;6(3):199-204.

Stroud S, Rodriguez. Arterial puncture and cannulation. In: Reichman EH, Simon RR. *Emergency Medicine Procedures*. 1st. New York: McGraw Hill; 2003:398-410.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 2

Brain Tissue Oxygen Monitoring

M. Kamran Athar, MD[1] and Jawad F. Kirmani, MD[2*]
[1]Neuro- Critical Care, Thomas Jefferson University,
Philadelphia, PA, US
[2] New Jersey Neuro Science Institute, JFK Medical Center,
Edison, NJ, US

Introduction

Measurement of brain tissue oxygen tension (PbO_2) is being increasingly used as a monitoring modality in the neurological intensive care unit. It can be measured continuously with a small flexible micro-catheter that is inserted into the brain parenchyma.

PbO_2 serves as a marker of the balance between regional oxygen delivery and consumption.

Indications

Brain tissue oxygen monitoring is most useful in brain injury with suspected regional ischemia such as the following:

- Severe traumatic brain injury (TBI) with poor Glasgow Coma Scale (GCS) score of 3-8 and an abnormal computed tomography (CT) head scan (Level III recommendation of the Brain Trauma Foundation)
- Poor grade subarachnoid hemorrhage (SAH) – Hunt & Hess Grade 3 or greater
- Large hemispheric infarctions
- During and after cerebrovascular surgery

* Email: jkirmani@gmail.com

Contraindications

- Coagulopathy
- Insertion site infections

Preparation

PbO_2 monitors are typically placed by neurosurgical staff in the operating room or at the bedside in the intensive care unit.

Procedure

The micro-catheter is inserted into brain parenchyma either through a bolt (a hollow-bore subarachnoid screw) inserted into the skull or through a craniotomy site and tunneled under the skin.

The micro-catheter should pass through the gray matter into the white matter.

It is placed *near* the injured brain tissue, directly avoiding areas of infarct or hematoma. Micro-catheters are approximately 0.5 mm in diameter and the measured tissue volume is 17 mm^3.

Once the probes are in place, the cables are connected to the monitor and the system is calibrated with a smart card. A "run in" or equilibration time of up to 30 minutes is required for the brain tissue to stabilize from the probe insertion, following which the PbO_2 readings are reliable. A CT scan of the head should be performed after insertion to confirm the parenchymal probe positioning.

Complications

- There is a small amount of zero-drift (1.5 mmHg) and sensitivity-drift (-8.5%) over time
- Hematoma
- Infection
- Catheter dislodgement
- Compared to ICP monitors, PbO_2 monitoring devices are less prone to accidental displacement and are unaffected by head movement

Interpretation

PbO_2 represents the partial pressure of oxygen in the extracellular fluid in the brain. It is the balance between oxygen availability and oxygen consumption. PbO_2 can be used as an

endpoint for optimizing cerebral perfusion pressure (CPP). It can be also used as an outcome predictor following brain injury. Multiple hypoxic thresholds have been evaluated which allow for multiple definitions of hypoxia. The most commonly used threshold is PbO_2 <10 mmHg with a normal range being between 15-30 mmHg.

References

De Georgia MA, Deogaonkar A. Multimodal monitoring in the neurological intensive care unit. *Neurologist*. 2005;11(1):45–54.

Kett-White R, Hutchinson PJ, Al-Rawi PG, et al. Adverse cerebral events detected after subarachnoid hemorrhage using brain oxygen and microdialysis probes. *Neurosurgery*. 2002;50(6):1213–1221; discussion 1221–1222.

Kiening KL, Unterberg AW, Bardt TF, Schneider GH, Lanksch WR. Monitoring of cerebral oxygenation in patients with severe head injuries: brain tissue PO2 versus jugular vein oxygen saturation. *J. Neurosurg*. 1996;85(5):751–757.

Nortje J, Gupta AK. The role of tissue oxygen monitoring in patients with acute brain injury. *Br. J Anaesth*. 2006;97(1):95–106.

van den Brink WA, van Santbrink H, Steyerberg EW, et al. Brain oxygen tension in severe head injury. *Neurosurgery*. 2000;46(4):868–876; discussion 876–878.

Vidgeon SD, Strong AJ. Multimodal cerebral monitoring in traumatic brain injury. *JICS* 2011; 12 (2): 126–133.

Wartenberg KE, Schmidt JM, Mayer SA. Multimodality monitoring in neurocritical care. *Crit. Care Clin*. 2007;23(3):507–538.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 3

Bronchoscopy

Ganga Ranasuriya, MD[1], Rohan Arya, MD[1], Adrian Pristas, MD[3] and Archana Mishra, MD, MS[2*]
[1]Cooper Medical School of Rowan University, Camden, NJ, US
[2]State University of New York at Buffalo, Buffalo, NY, US[*]
[3]Bay Shore Hospital at Meridian Health, Holmdel, NJ, US

Introduction

Bronchoscopy is a procedure that involves the use of a fiber-optic or video bronchoscope to directly visualize the trachea and bronchial tree in real time. There are two types; flexible and rigid bronchoscopes. Flexible bronchoscopy is the most commonly performed in the intensive care unit (ICU) and it is used both as a diagnostic and therapeutic procedure. Rigid bronchoscopy is not usually performed in the ICU and is beyond the scope of this chapter. Bronchoscopy in the ICU setting is usually performed on patients that are intubated and have a stable airway.

We will assume this for this chapter. The flexible bronchoscope consists of a flexible sheath that contains cables that allow the tip of the bronchoscope to be flexed and extended, fiber-optic fibers for transmitting endobronchial images, a light source, and a working channel.

Indications

- Obtain broncho-alveolar lavage (BAL) fluid for cultures and cytology.
- Therapeutic cleaning (suctioning) of obstructed airways (mucous impaction, clots from previous bleed) that are causing significant atelectasis.
- Aiding in bedside percutaneous tracheostomy
- Difficult intubation

- Evaluation of vocal cord pathology
- Isolation of bleeding airway in hemoptysis.

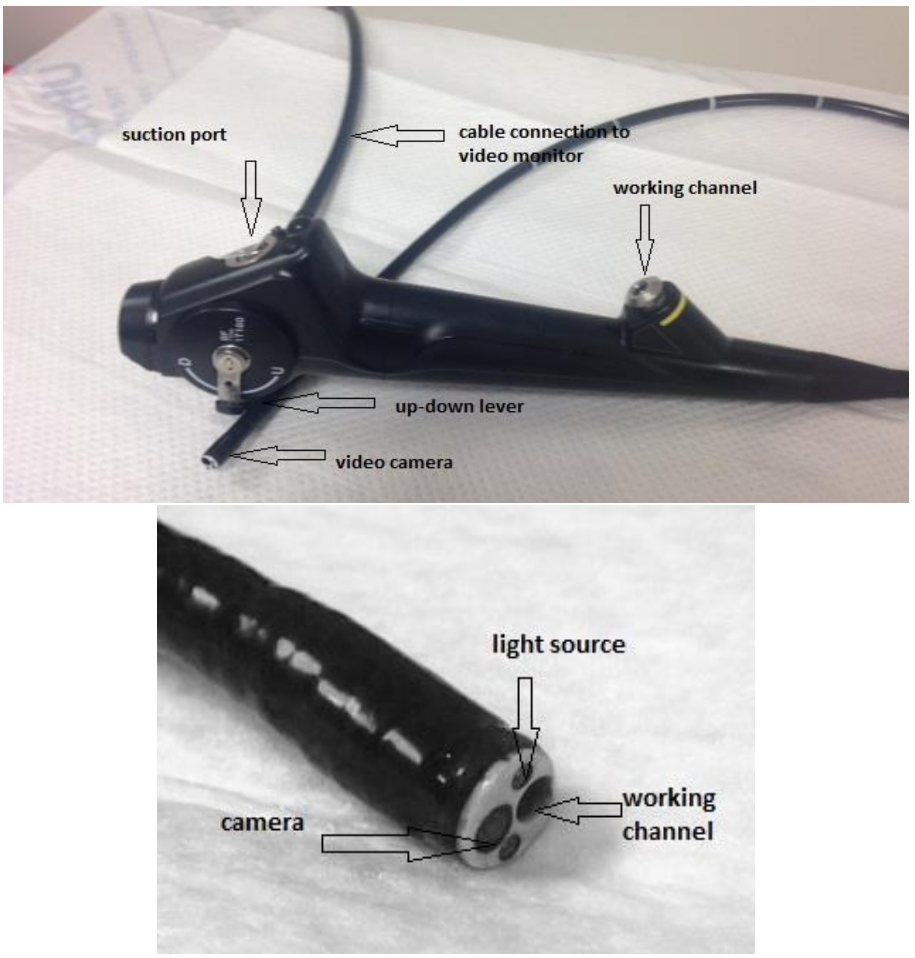


Figure 1.

Contraindications

- Active/ongoing coronary ischemia; this includes patients who have had an ischemic event with the last 6 weeks.
- Unstable congestive heart failure
- Patient with unstable/exacerbated airway disease: they are at risk for life threatening acute and life threatening bronchospasm.
- Severe hypoxemia:
    - Resting arterial oxygen tension (PaO2) less than 60 mmHg.
    - Oxy-hemoglobin saturation, also known as arterial saturation (SpO2) less than 90%.
- Hemodynamic instability.

Procedure

1. Determine the indication and ensure there are no contraindications.
2. Obtain Informed consent from patient or a appropriate surrogate.
    a. Prior to starting the procedure: Ensure FiO2 is set at 100%
    b. Place bite block to prevent the patient from biting down and damaging the bronchoscope.
    c. Have appropriate sedation available.
    d. ETT connecter piece has been placed; this is an adaptor that connects the ETT to the ventilator tubing but has an additional port to allow the bronchoscope to pass through it.
    e. Ensure the suction set up is functional.
    f. Review current imaging to determine target area; for example left lower lobe infiltrate for BAL.

3. After obtaining adequate sedation insert the bronchoscope through the mouth piece, push forward until you are out of the ETT and the trachea and main carina is visualized. At this point you can instill upto 60 ml of 1% lidocaine through the working channel to anesthetize the airways,
4. If the patient is not coughing and appears comfortable, begin to inspect the airways looking for endobronchial lesions or any other abnormalities. These should be inspected down to the first segments.
5. Once the airways have been inspected proceed to the target airway.
6. For BAL; connect the sputum trap to the suction port of the bronchoscope with one end connect to the scope and the other end to the suction. Wedge the end of the bronchoscope into the desired airway and instill 60 ml saline into the airway. After waiting 10-15 seconds suction of the instilled fluid by rapidly pressing down on the suction port. This can be repeated in several airways. Once an adequate volume of BAL fluid is obtained, disconnect the sputum trap apparatus and connect the suction tubing back to the bronchoscope.
7. For therapeutic airway suctioning: proceed with steps 1 to 7, once done with the BAL portion, return to each airway and instill 30-60 ml saline followed by aggressive suctioning to clean out the increased secretions.
8. 9. All obtained samples should be sent for quantitative cultures for acid fast bacillus (ABF), fungus and bacterial, white blood cell (WBC) count with differential and cytology. Special tests include silver stain for suspected PCP, hemosiderin laden macrophages for suspected alveolar hemorrhage or CHF and fat stains for suspected chronic aspiration.

Important Considerations

- If at any point the patient develops critical hypotension or bradycardia the bronchoscope should be withdrawn from the airway and the procedure should be stopped at least until the hemodynamics improve.

- For persistent coughing, one of a few maneuvers can be implemented- better anesthetization of the airway with 1% lidocaine, increased sedation or pulling back the bronchoscope to the carina until the coughing stops.
- If patient develops persistent hypoxemia see #1.

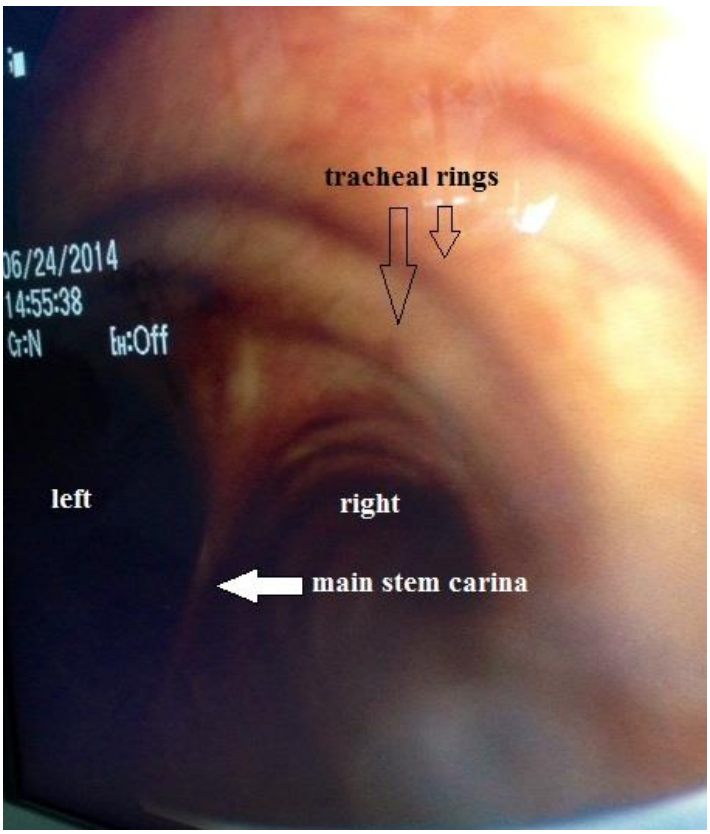


Figure 2. bronchoscopic view of the main stem carina.

References

Landa, Jose F. "Indications for bronchoscopy." *CHEST Journal* 73.5_Supplement (1978): 686-690.

Alamoudi, Omer S., et al. "Bronchoscopy, indications, safety and complications." *Saudi Medical Journal* 21.11 (2000): 1043-1047.

Kvale, Paul A. "Is it really safe to perform bronchoscopy after a recent acute myocardial infarct?." *CHEST Journal* 110.3 (1996): 591-592.

Shinnick, James P., Robert F. Johnston, and Theodore Oslick. "Bronchoscopy during mechanical ventilation using the fiberscope." *CHEST Journal* 65.6 (1974): 613-615.

Liebler, Janice M., and Catherine J. Markin. "Fiberoptic bronchoscopy for diagnosis and treatment." *Critical care clinics* 16.1 (2000): 83-100.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 4

Central Venous Catheter Placement

Ganga Ranasuriya, MD[1], Carol H. Choe, MD[1],
Alaaeldin Soliman, MD[2]
and Sri Sujanthy Rajaram, MD, MPH[*2]
[1] Cooper Medical School of Rowan University, Camden, NJ, US
[2] Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Central venous catheters (CVCs) are placed frequently in the Intensive Care Unit (ICU). Catheters may be placed in the femoral vein, subclavian vein or internal jugular vein (IJ). The most commonly used CVCs in the ICU are temporary, non-tunneled catheters. It is important to determine which CVC to use:

- Single lumen introducer sheath – used for rapid, large volume administration of fluids and blood products. It is also used to place other catheters such as pulmonary artery catheters and transvenous pacemakers.
- Triple lumen catheter – with three functioning ports, this catheter allows for the simultaneous administration of multiple medications, nutrition, and fluids.
- Dialysis catheter – for emergent, temporary hemodialysis in critically ill patients.
- Central Venous Oxygen monitoring catheter (Precept catheter)- A fourth white port allows continuous monitoring of central venous oxygen saturation in addition to the three ports of a triple lumen catheter.

[*] Email: sujanty@gmail.com

Indications

- Medication and vasopressor administration
- Hemodynamic monitoring and resuscitation
- Difficult peripheral IV access.
- Transvenous pacemaker placement.
- Central Venous Pressure monitoring, Central venous oxygen saturation monitoing
- Requirement for continuous renal replacement therapy (CRRT), plasma exchange, plasmapheresis.
- Total Parenteral nutrition administration
- Hypothermia Catheters: Used only at femoral site for intravascular cooling. Two infusion ports for cold saline and two ports for central access

Contraindications (Relative)

- Coagulopathy, particularly if accessing the subclavian site.
- If pacemakers are present avoid the same side

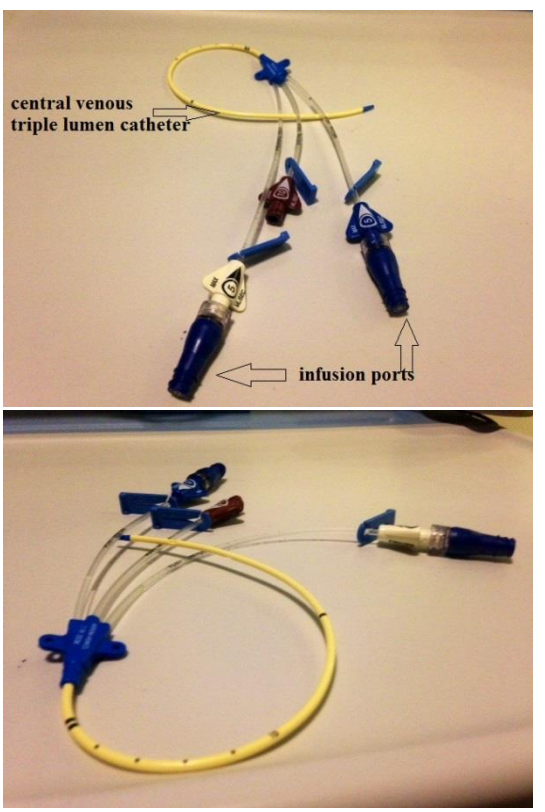


Figure 1. Triple lumen central venous catheter.

Preparation

Positioning

- Once the access site and approach are chosen, it is important to position the patient to provide maximal comfort to the operator and to expose the vein appropriately. To avoid air embolism and to allow maximum venous filling in the internal jugular and subclavian veins, the patient may be placed in Trendelenburg position. Some patients with respiratory difficulties may find this challenging and should use supine position.
- Premedication with opiates and or benzodiazipines is helpful in awake patients

Ultrasound

- The use of ultrasound (US) is now the standard of care when placing a CVC to better visualize the venous site of interest, view anatomic variability, and identify correct placement of the catheter in the target vessel. The ultrasound probe should be covered with a sterile ultrasound cover when used. Veins are compressible and artery is noncompressible and pulsatile.

Procedure

1) Prepare and drape the patient using aseptic technique.
   a. Chlorhexidine-based cleaning solutions are commonly available in sterile draping kits. Thoroughly cleansing the area decreases the incidence of infection. When a site on the upper body is chosen, consider cleaning the adjacent area on the chest or neck in the event an alternative site is required during the procedure.
   b. To reduce infectious complications, all central venous access procedures should be performed with full barrier precautions including sterile drapes large enough to cover the entire patient, surgical antiseptic hand wash, sterile gown, sterile gloves, mask, and cap.
2) Infiltrate the skin with local anesthetic (1% or 2% Lidocaine).
3) Saline-lock the port(s) of the catheter with sterile saline solution. If there is more than one port, the most distal port site is clamped until the guidewire exits. All other ports may either be clamped or saline-locked with sterile caps.
4) With ultrasound guidance (Veins are compressible and arteries have pulsatile flow with ultrasound. If using Doppler ultrasound arteries are dopplerable) place the finder needle in the vein and advance the needle in the appropriate direction with negative traction on the syringe. This will allow for immediate blood return once the vessel is accessed. Once venous blood return is seen in the syringe, disconnect the syringe from the needle. Maintain a good hold on the needle to prevent accidental through-and-through puncture of the vessel.
5) The guidewire is then introduced through the needle lumen. US can again be used to confirm location of the guidewire. Once the guidewire is in the vessel, the needle is

removed while the operator maintains a firm grip on the guidewire. Always maintain possession of the guidewire throughout the entire procedure.

6) Make a small skin incision at the guidewire entry site.
7) Using Seldinger technique, thread the dilator over the guidewire. Advance the dilator through the tissue layers using a slow twisting motion. It may be necessary to enlarge the skin incision if it is insufficient to pass the dilator with ease. Once the tissue layers have been appropriately dilated, remove the dilator.
8) Thread the catheter over the guidewire, till the tip is about an inch before the wire skin-entry site, hold the catheter with one hand, with the other hand, pull the wire out while threading the catheter in until one inch of wire comes out of the brown port (or the proximal port for other catheters), hold the visible wire outside the brown port, then advance both the catheter and the wire to the desired length and pull the wire out. Or hold the wire at the tip of the brown port firmly and advance the catheter only to the desired length.
9) Remove the guidewire (Never loose the guidewire inside).
10) Using a sterile saline-filled syringe, aspirate blood from each port to ensure good blood return. Flush all ports with saline, taking care not to introduce air into the patient.
11) Suture the catheter in place and obtain a post-procedure chest xray to confirm appropriate placement, not needed for femoral line placement.
12) Use heparin for dialysis catheters in each port as directed in the port to prevent clotting if no contraindications for heparin (usually 1.5 cc heparin in each port)

Sites

1. **Internal Jugular (IJ) Vein Line placement:** Preferrably placed under ultrasound guidance. Advantages of the IJ site includes lower risk of pneumothorax, ease of compressibility of the vessel in the event of bleeding or arterial puncture and a straight path from the right IJ to the superior vena cava make it as the preferred site particularly for dialysis catheters. Disadvantages are difficult anatomical location in obese or edematous patients and less comfortable and more difficult to keep it clean in intubated patients.

   Median Approch: Commonly used approach. Turn the head to the contralateral side, Identify sternal and clavicular heads of the sternocleidomastold, if those are not apparent, flex the neck against resistance and insert needle just inferior to the junction of the two heads and advance toward the ipsilateral nipple at a 30-45° angle and IJ vein should be reached within 3cm.

   Anterior approach: Feel the carotid pulse in the triangle, insert the needle at 30-45° angle lateral to the carotid pulse, along the medial edge of the sternocleidomastoid, almost at the inferior margin of the thyroid cartilage

   Posterior or Lateral approach: Insert needle in the midlle and below the clavicular head of the sternocleidomastoid (posterior lateral margin) about a few centimeters above the sternoclavicular joint and direct the needle towards the contralateral nipple at a 10° to 15° angle

2. **Subclavian Vein (SC) line placement:** Ultrasound is generally not useful in SC lines. Preferred first line of choice due to easily identified bony landmarks, improved patient comfort, ease of dressing to keep it clean and lower incidence of infection. It has higher risk for pneumothorax than IJ. Avoid SC puncture in coagulopathy patients because it is a non compressible site.

Insert the needle just below the clavicle and aim towards the sternal notch or towards the head. Needle should never be aimed more than 10-15 degree angle from the skin. Try to hit the clavicle with the needle after adequate local anesthesia and press the needle down to get under the clavicle aiming towards the sternal notch.

Lateral approach: Needle insertion is at the medial 2/3 and lateral 1/3 of the clavicle. This approach has a high risk for arterial puncture.

Middle approach: Needle is inserted just below the middle of the clavicle

Medial approach: Needle is inserted at the medial 1/3 and lateral 2/3 of the clavicle. Useful in obese patients.

Supraclavicular approach is occasionally used and ultrasound may be helpful in this approach.

3. **Femoral Vein:** Identify the femoral triangle and feel the femoral pulse or use the ultrasound. Insert the needle medial to the pulsation just below the inguinal ligament. Infection rates are high at this site.

Length of Insertion

Length of insertion: 14 to 16 cm from right sided IJ or SC, 16 to 18 cm from left sided IJ insertions. IJ and SC veins should not be inserted to a depth of 20 cm. Dialysis catheters the entire length needs to be inserted and secured at the skin, hence 20 cm catheters should not be used at the IJ or SC sites.

- In children measure the length from 2nd rib to entry site and insert to the measured length only. Use 3Fr. catheter for infants, 4Fr. for toddlers, 5Fr. for children and 7Fr. For adolescents. Cleaning is with chloroxidine for children over 2 months of age. Use 18, 22 or 25 gage needle or central venous catheter kit. Use supplemental oxygen, pulse oximeter and EKG monitor all the time and have resuscitation cart available.

Troubleshooting

1. After placement, Am I in the artery? If in doubt, checking blood gas and CXR will take time.
   - Connect the brown port to a pressure transducer and check for wave form and pressure.

- Or use ultrasound to see if the vessel is compressible or not.

2. Wire does not advance beyond the needle length:
   - Make sure you are still in the vein by having good blood flow
   - Retrieve the wire about a centimeter, twist the wire to 180 degrees to change the direction of the wire j-tip and advance
   - If still does not advance, keep the wire in the vein, pull the metal needle out, advance the plastic angiocatheter over the wire into the vein, retrieve the wire out back to the original plastic hub, check blood flow through the angiocatheter, readvance the wire j-tip in different direction until wire advances then remove angiocatheter and proceed with CVC placement.
3. Blood is not coming out in one port before flushing: Most likely the hole of the port is against the vein wall. Push 1 cc of normal saline in the lumen and look for blood flow.
4. IJ Insertion site is bleeding despite compression: Most likely because the dilator created a hole larger than the catheter caliber. Turn patient's head towards the shoulder to avoid over stretching of the vein.
5. Sutures are too tight during removal: Cut the plastic rim, slide it out of the suture then will have room to advance scissor tip to cut the sutures.

Complications

- Pneumothorax -Encountered with internal jugular vein and subclavian vein access attempts.
- Malposition
- Hemothorax
- Bleeding
- Infection- Femoral site is higher than IJ and lowest is SC
- Carotid arterial puncture
- Thoracic duct injury (with left subclavian or internal jugular approach)
- Air embolism- Serious and underecognized complication. Airembolism can happen during insertion or during catheter removal. Catheter removal must be done in supine position and during exhalation in spontaneously breathing patients. Large amount of air can get into the IJ or SC vein and can cause right ventricular out flow tract obstruction leads to cardiac arrest. Patient should be resuscitated in left lateral position in order to allow the air to disseminate to the pulmonary cirlcualtion.

Peripherally Inserted Central Venous Catheter (PICC)

PICC lines are inserted under ultrasound guidance though cephalic, basilic or brachial veins. After direct needle entry without using a syringe, guide wire is inserted and the introducer is passed through the guidewire. Catheter is positioned and secured at the measured length from the site of entry to the right atriam. PICC lines have high rates of

malposition and thrombosis and associated with frequent displacement of the catheter with arm movement.

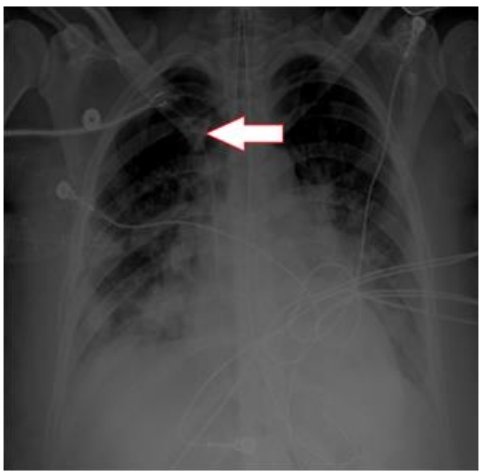


Figure 1. subclavian vein central catheter placement.

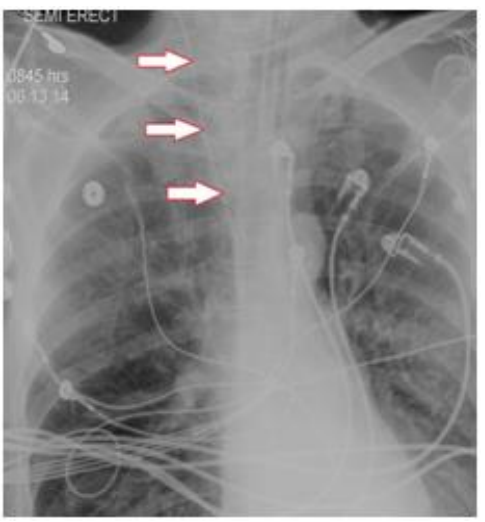


Figure 2. Right internal jugular placement.

References

Burden A., MD; Torjman MC., PhD.; Dy G., BS.; Jaffe J., DO.; Littman JJ., MD.; Nawar F., MD.; Rajaram SS., MD.; Schorr C., RN, MSN.; Staman G., RN.; Reboli A., MD. Prevention of central venous catheter-related bloodstream infections: is it time to add simulation training to the prevention bundle. *Journal of Clinical Anesthesia* 2012

Durrani Q., MD; Gajera M., MD; Punjabi V.,MD; Shastri G, MD., Rajaram SS.M.D, Incidence of PICC line associated thrombosis in patients already on prophylaxis for thromboembolism. *Critical Care Med* 2009; 37(Suppl.):A365.

Graham AS, Ozment C, Tegtmeyer K, Lai S, Braner DAV. Videos in clinical medicine. Central venous catheterization. N. *Engl. J. Med.* 2007;356(21):e21.

Rajaram SS, Dellinger RP, Positioning for central venous access. Seminars in Anesthesia, *Perioperative Medicine and Pain* (Elsevier) 2005; 24:211-213

www.uptodate.com , central venous catheterization

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 5

Cricothyroidotomy

Emily Damuth, MD[1] and Sri Sujanthy Rajaram, MD, MPH[2]
[1]Cooper University Hospital, Camden, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Prior to attempting intubation, the Intensivist must evaluate patients for indicators of difficult intubation. This allows careful planning of alternatives and verification that necessary equipment is available in the event a difficult airway is encountered. Cricothyroidotomy is an emergent surgical procedure typically used as a last resort in the management of a failed airway in a patient whom the physician cannot intubate and cannot ventilate.

Indications

Cricothyrotomy is an emergent advanced airway procedure reserved for patients in whom oral or nasotracheal intubation is unsuccessful or contraindicated and the establishment of a definitive airway is necessary to avoid life-threatening hypoxemia or hypoventilation. Patients with airway obstruction secondary to tumor, hemorrhage, trauma or congenital deformity are more likely to require an emergent surgical airway.

Contraindications

There are no absolute contraindications to cricothyrotomy. Relative contraindications include age less than 10 years old, tracheal transection or laryngeal fracture. Needle

cricothyrotomy is preferred in children less than 10 years old due to the risk of damage to the small cricothyroid membrane and lack of laryngeal prominence.

Preparation

At minimum, required equipment includes, scalpel (10 or 11-blade); 6-mm endotracheal tube (ETT) with stylet or 5-mm tracheostomy tube; 10-cc syringe; end-tidal CO_2 detector; bag valve mask (BVM); oxygen source and suction. Sterile protective equipment and 1% lidocaine with epinephrine should be utilized when time allows. A tracheal dilator, tracheal hook, gum elastic bougie and a curved hemostat can be used if available, but are not essential.

Be familiar with the anatomy of the neck. In the middle of the neck, anteriorly hyoid bone is the upper most palpable structure. Hyo-thyroid membrane connects the hyoid bone with the large palpable thyroid cartilage. Thyroid cartilage is connected with the ring like palpable cricoid cartilage through crioco-thyroid ligament. Below cricoid cartilage are the tracheal rings. Cricothyroidotomy is an establishment of an airway through crioc-thyroid ligament.

Procedure

Place the patient in a supine position with the neck extended, unless cervical spine precautions must be maintained. Preoxygenate via BVM as the patient's condition allows. Stabilize the cricothyroid membrane, located between the thyroid cartilage above and cricoid cartilage below, with the non-dominant hand. Apply iodine, if time permits. Make a midline *vertical* skin incision at the level of the cricothyroid membrane. A vertical skin incision is preferred to prevent recurrent laryngeal nerve injury and allow extension to ensure appropriate position. While continuing to stabilize the larynx with the thumb and middle finger of the nondominant hand, make a *horizontal* incision through the cricothyroid membrane.

After accessing the trachea with the blade, use a finger to bluntly widen the cricothyroid membrane opening. This can also be performed with the blunt end of the scalpel, but potentially risks injury to the provider. Always maintain contact through the tracheal opening with either a finger or tracheal hook to avoid displacing the airway and creating false tract. If available, a dilator may be used at this stage to widen the opening. Insert a tracheostomy tube or pass the endotracheal tube (with stylet in place) to a depth of 2 to 3 cm. Inserting an ETT beyond this distance risks right mainstem intubation. Alternatively, insert a gum elastic bougie through the incision and pass the endotracheal tube over the bougie and into the trachea. Inflate cuff and confirm placement with end-tidal CO_2 ($ETCO_2$) and auscultation of bilateral breath sounds. Suture the tracheostomy or endotracheal tube in place and obtain chest x-ray for confirmation of placement. Surgery should be consulted for definitive tracheostomy placement within 72 hours to prevent subglottic stenosis.

Complications

Esophageal injury or perforation, bleeding, infection and subcutaneous emphysema can result from cricothyrotomy.

References

Hill, C; Reardon, R; Joing, S; et al. Cricothyrotomy technique using gum elastic bougie is faster than standard technique: a study of emergency medicine residents and medical students in an animal lab. *Academic Emergency Med*, 2010, 17(6), 666-669.

Hsiao, J; Pacheco-Fowler, V. Videos in Clinical Medicine: Cricothyroidotomy. *NEJM*, 2008, 358(22), e25.

Smith, MD. (2011). Surgical Airway Management (Tintinalli JE, Ed). In *Tintinalli's Emergency Medicine: A Comprehensive Study Guide* (209-215). New York: The McGraw Hill Companies, I.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 6

Chest Tube (Tube Thoracostomy) Placement

Rohan Arya MD[2] and Sri Sujanthy Rajaram MD, MPH[1*]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US

Introduction

Chest tube (thoracostomy) placement involves the placing of a sterile drainage catheter through the chest wall and into the pleural space. It can be performed at the bedside and in the operating room after thoracic surgery. The placement of such a catheter is to drain the pleural space of either air (pneumothorax) or fluid (pleural effusion, hemothorax, para-pneumonic effusion or empyema). The reason for drainage of this fluid can be for treatment of dyspnea, improvement of lung function or source control for infections.

Indications

- Pneumothorax
- Pleural effusion
- Hemothorax
- Pleurodesis
- Sclerosing agents can be instilled or insufflated as treatment for recurrent effusions.

* Email: sujanty@gmail.com

Contraindications

- Coagulopathy: patients on anti-coagulation, with thrombocytopenia or bleeding diasthesis. The risk of placing a chest tube in a coagulopathic patient may be overlooked when presented with life threatening situations such as a tension pneumothorax.
- Hepato-hydrothorax: patients with cirrhosis and consequent transudative pleural effusions should not be managed with chest tube placement.

Preparation

Establish the indication for the chest tube: pneumothorax, pleural effusion, hemothorax, empyema. The indication for thoracostomy will dictate the anatomical location of chest tube placement as well as chest tube size and type.

Evaluate for contraindications and need for prophylactic antibiotics (penetrating injuries).

Thoracostomy trays usually come in complete sets that contain the chest tube, local anesthetic, sterile drape and tools that will be used during the procedure.

Materials needed

- Chest tube kit (standard vs Seldinger technique).
  - ✓ 1% lidocaine
  - ✓ 25 gauge needle
  - ✓ Sterile gloves cap and gown.
  - ✓ Suture
  - ✓ Sterile dressing
- Surgical chest tube (typically for hemothorax and empyema)
- Water seal drainage system

Procedure

Determine the size of chest tube needed (Table 1). Pleural fluid tends to collect in dependent areas of the chest cavity; air rises to non-dependent areas. The patient must be positioned either sitting up or semi-reclined.

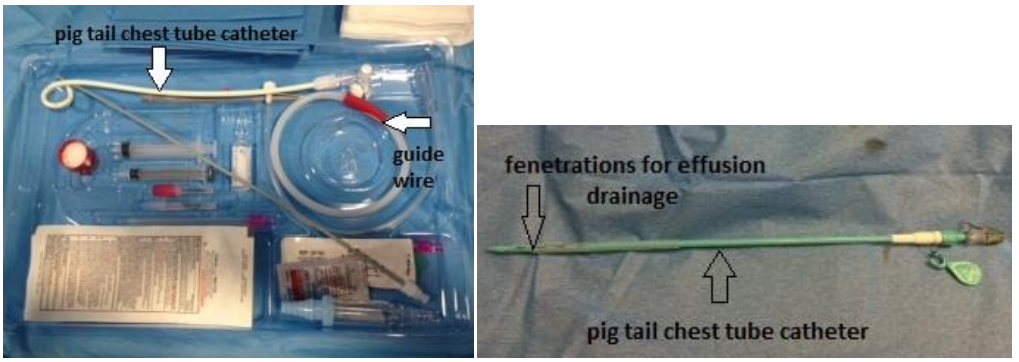
Seldinger Technique

1. For a pneumothorax the pigtail chest tube or a 12 french small tube can be placed in the 2nd intercostal space in the mid-clavicular line. For pleural effusions, ultrasound of the chest should be done to evaluate for appropriate placement of the chest tube. The site should be marked at the time of the chest ultrasound.

2. The area that is marked for chest tube placement should be cleaned and sterilely prepped. The entire procedure should be performed under standard sterile precautions.
3. Using the 25-gauge needle and lidocaine, create a wheal over the proposed insertion site. While maintaining negative pressure in the syringe, continue to attempt to anesthetize the proposed tissue tract down to the pleura. Stop when fluid or air is aspirated. Note the depth of the needle and then remove it.
4. While maintaining negative pressure, insert the larger introducer needle into the same anesthetized tract. Once fluid or air is aspirated, feed the guide wire into the pleural space, through the introducer needle.
5. Remove the introducer needle and dilate the tract by passing the dilator over the guide wire into the pleural space.
6. Remove the dilator and pass the chest tube over the guide wire into the pleural space.
7. Remove the guide wire. Attach a 3-way stopcock to the chest tube.
8. Secure the chest tube to the chest by placing a purse string suture around the chest tube insertion site.
9. Connect the chest tube to the water seal drainage system and place on low wall suction. Typically negative pressure suction of 20 cm H2O is used.
10. Order a CXR to confirm chest tube placement and to re-evaluate the pneumothorax or pleural effusion.

Table 1. Chest tube sizes for various indications

Indication	Size	Technique	Comments
Broncho-pleural fistula	20-28	Seldinger-standard	These manifest as large air leaks on patients on mechanical ventilation.
Tension pneumothorax	14-36	Needle decompression then Seldinger	If unstable will need immediate decompression. If stable, elective placement is preferred.
Pleural effusion (transudate or malignancy)	14-16	Seldinger	Adequate drainage can usually be obtained with smaller bore tubes.
Parapneumonic	14-24	Seldinger	Drainage with smaller bore is preferred. If drainage fluid is more viscous, a larger bore tube may be needed.
Empyema	16-28	Seldinger- standard	Smaller tubes will likely become occluded because of viscous fluid and / or debris.
Hemothorax	18-40	Seldinger- standard	Smaller tubes are likely to clot.



Pig tail chest tube catheter kit.

Standard technique (for larger bore chest tubes, 24 Fr or greater)

1. For a pneumothorax, if using a surgical chest tube, it should be placed in the 4th intercostal space along the anterior axillary line. For an empyema or hemothorax, tube placement should be done in the 5th or 6th intercostals space along the mid-axillary line.
2. The area that is marked for chest tube placement should be cleaned and sterilely prepped. The entire procedure should be performed under standard sterile precautions.
3. If the patient is supine, raise the ipsilateral arm and place over the head to expose the rib space. If the patient is in the lateral decubitus position, place the ipsilateral arm on a bedside tray table.
4. Using a 25-gauge needle and lidocaine, create a wheal over the center of the rib. While maintaining negative pressure in the syringe, gently work over the top of the rib and continue to anesthetize the skin, intercostal muscles, and periosteum of the rib. Continue to anesthetize the proposed tissue tract down to the pleura. Stop when fluid or air is aspirated.
5. Slowly retract the needle while aspirating fluid or air until it stops, then inject more lidocaine to numb the pleura.
6. Using a scalpel, make a 2 to 3 cm transverse incision over the center of the rib. Then, using a blunt-tipped clamp, dissect over the top of the rib and make a subcutaneous tunnel down to the pleura.
7. Carefully puncture the parietal pleura with a hemostat. A rush of air (pneumothorax) or fluid (effusion) may be seen.
8. Insert a gloved finger into the pleural space to clear any debris and to evaluate for any lung injury that may have been caused by the tip of the hemostat.
9. Using the gloved finger as a guide, introduce the chest tube into the pleural space. Ensure that the most distal hole of the chest tube is inserted into the chest cavity.
10. Secure the chest tube with a purse string suture, leaving an equal length of suture on each side. Using the two ends, criss-cross the suture around the tube in a Roman sandal fashion. This should be tight enough to cause slight dimpling of the tube. Additional simple sutures may be required on either end of the tube to keep the incision site closed.

11. Place a clean dressing that contains Vaseline gauze over the chest tube insertion site (optional).
12. Connect the chest tube to the water seal drainage system and place onto low wall suction.
13. Follow up with CXR fro correct tube placement and expansion of the lung or drainage of fluid.

Complications

- Infection
- Bleeding
- Lung parenchyma damage
- Subcutaneous emphysema
- Persistent pneumothorax or hemothorax
- Incorrect tube placement
- Subdiaphragmatic tube placement
- Cardiac arrhythmias
- Brocho-Pleural Fistula: If persistent air-leak is present suspect a fistula. When tube throcostomy is placed for a pneumothorax, after complete expansion of the lung return of airleak may indicate a broncho-pleural fistula. In such cases chest tube should not be removed until the air-leak stops and the fistula heals, particularly if the patient is on the ventilator.

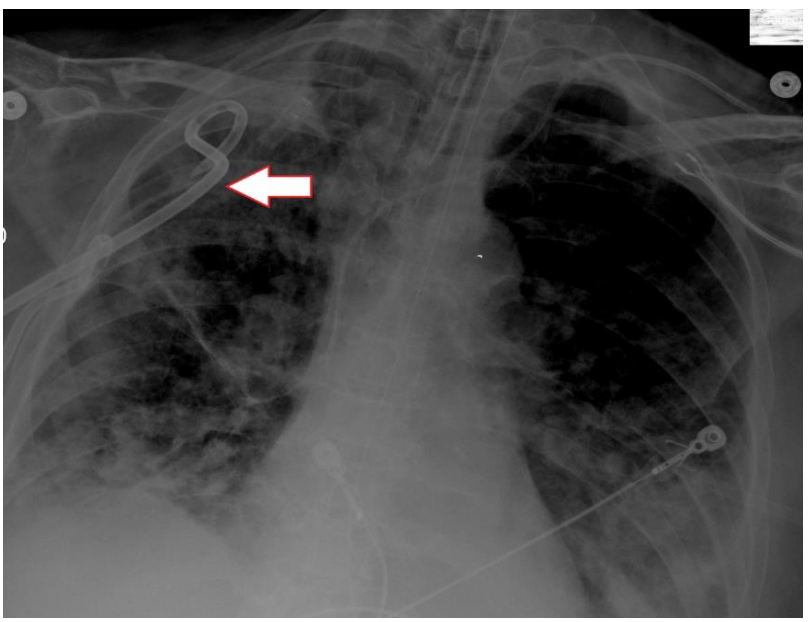


Figure 1. Anterior pigtail chest tube catheter placement for pneumothorax.

References

Canto A, Blasco E, Casillas M, Zarza A, Padilla J, Pastor J, et al. Thoracoscopy in the diagnosis of pleural effusion. *Thorax*. 1977;32(5):550-4.

Casal RF, Eapen GA, Morice RC, Jimenez CA. Medical thoracoscopy. *Current opinion in pulmonary medicine*. 2009;15(4):313-20.

Grogan DR, Irwin RS, Channick R, Raptopoulos V, Curley FJ, Bartter T, et al. Complications associated with thoracentesis: a prospective, randomized study comparing three different methods. *Archives of internal medicine.* 1990;150(4):873-7.

http://www.uptodate.com/contents/an-overview-of-medical-thoracoscopy

Laws D, Neville E, Duffy J. BTS guidelines for the insertion of a chest drain. *Thorax.* 2003;58(suppl 2):ii53-ii9.

Light RW. Pleural effusions. *Medical Clinics of North America*. 2011;95(6):1055-70.

Menzies R, Charbonneau M. Thoracoscopy for the diagnosis of pleural disease. *Annals of internal medicine.* 1991;114(4):271-6.

Rahman NM, Ali NJ, Brown G, Chapman SJ, Davies RJ, Downer NJ, et al. Local anaesthetic thoracoscopy: British Thoracic Society pleural disease guideline 2010. *Thorax.* 2010;65(Suppl 2):ii54-ii60.

Rodriguez-Panadero F, Janssen J, Astoul P. Thoracoscopy: general overview and place in the diagnosis and management of pleural effusion. *European Respiratory Journal.* 2006;28(2):409-22.

Sokolowski Jr JW, Burgher LW, Jones Jr FL, Patterson JR, Selecky PA. Guidelines for thoracentesis and needle biopsy of the pleura. *American Review of Respiratory Disease.* 1989;140(1):257-8.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 7

Esophageal Balloon Tamponade (Sengstaken-Blakemore tube placement)

Pedro Dammert, MD[1], Rohan Arya, MD[2], and Sri Sujanthy Rajaram, MD, MPH[3,*]
[1]Kent General Hospital, Dover, DE, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US
[3]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Insertion of an esophageal and gastric tube with inflatable balloons can be employed to control upper GI bleeding from esophageal or gastric varices. Confirmation of variceal bleeding should be confirmed prior to placement of the balloon.

The tube most often employed for variceal tamponade is the Sengstaken-Blakemore tube. This has an esophageal balloon, a gastric balloon and a single gastric suction port.

Other options include the Minnesota tube, which has an additional esophageal suction port above the esophageal balloon. The Linton-Nachlas tube has a single gastric balloon.

Indications

- Temporary control of life-threatening refractory bleeding from esophageal or gastric varices.

[*] Email: sujanty@gmail.com.

Contraindications

- Recent esophageal surgery
- Hiatal hernia
- Esophageal diverticulum
- Relative risks
- History of esophageal radiation due to potential risk of rupture with dilation

Preparation

Equipment

- Balloon tamponade tube that has been cooled to make it stiff
- Lubricant
- 50 mL enteral syringe
- Two clamps
- Pulley system and orthopedic rope, or football helmet
- Sphygmomanometer
- Nasogastric tube
- Two wall suction systems

Procedure

1) Inflate balloons with air and test for leaks; deflate balloons.
2) Lubricate tube.
3) Insert the tube through the mouth for intubated patients and through the nare for non-intubated patients. The tube should be inserted up to 50 cm in length. Confirm placement by listening for gastric sounds.
4) It may be necessary to use direct laryngoscopy to advance the balloon into the esophagus under direct visualization.
5) Macgill forceps can be used to advance the balloon into the esophagus if necessary.
6) Initially inflate the gastric balloon with 50 mL of air and clamp it.
7) Obtain a portable abdominal x-ray to confirm the balloon is in the stomach, below the diaphragm.
8) If the position is confirmed, inflate the gastric balloon to 300 mL and clamp it.
9) Withdraw the balloon gently until resistance is felt. This resistance indicates that the gastric balloon is positioned against the patient's gastro-esophageal junction.
10) Securely fasten the tube to either a pulley device with 500 to 1000 mL of weight or taped to a football helmet to maintain tension on the tube.
11) If bleeding does not stop despite inflation of the gastric balloon, inflate the esophageal balloon to 30 to 45 mmHg with the sphygmomanometer.

12) Place a nasogastric tube at the level of the esophagus if the esophageal balloon is not inflated, or above the esophageal balloon if it is inflated. The purpose of this tube is to monitor for esophageal bleeding.
13) Place the gastric port and nasogastric tube to suction.
14) Obtain a second abdominal x-ray to confirm position.
15) All three ports are secured through a helmet placed on the patient's head and positioned securely.

Complications

- Esophageal rupture (8% of cases)
- Gastric rupture
- Mucosal pressure necrosis
- Aspiration pneumonia
- Balloon misplacement

Interpretation/Management

Balloon Management

- Esophageal balloon: Once the bleeding is controlled, reduce the pressure by 5 mmHg to a goal pressure of 25 mmHg. If bleeding resumes, increase the pressure by 5 mmHg. If the bleeding continues to be controlled, maintain the balloon pressure at 25 mmHg for 12 to 24 hours. Check the pressure every hour and deflate the balloon for a few minutes every 8 hours to assess for re-bleeding and prevent pressure necrosis.
- Gastric balloon: Deflate the balloon for a few minutes every 12 hours to assess for re-bleeding and pressure necrosis.
- The tube can be left in place for a total of 24 to 48 hours. Deflate the esophageal balloon at 24 hours if bleeding has stopped. Keep the gastric balloon in place for another 24 hours. If bleeding has stopped by then, remove the tube.

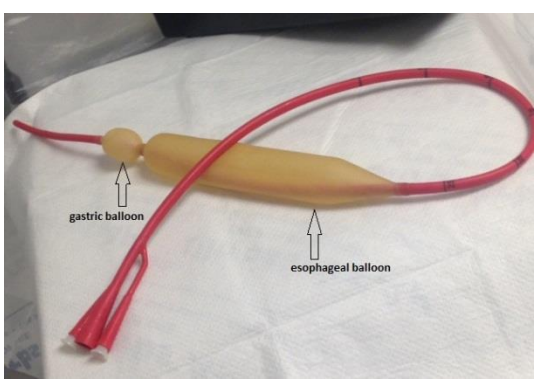


Figure 1(a). (Continued).

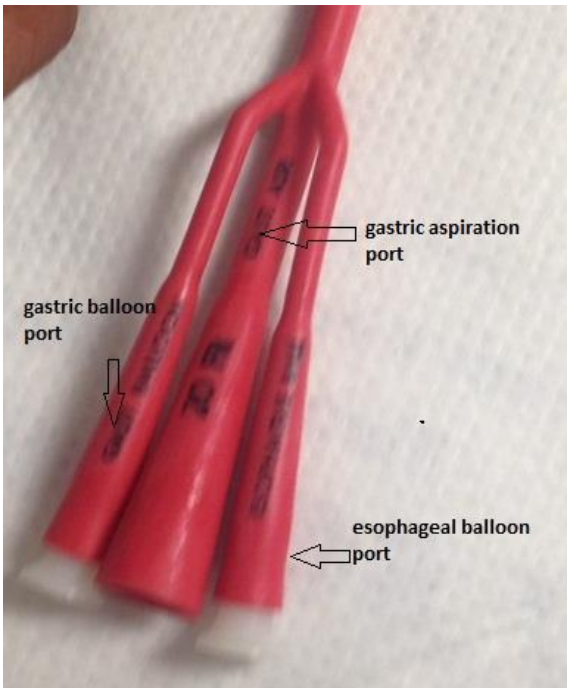


Figure 1(b). Sengstaken-Blakemore tube.

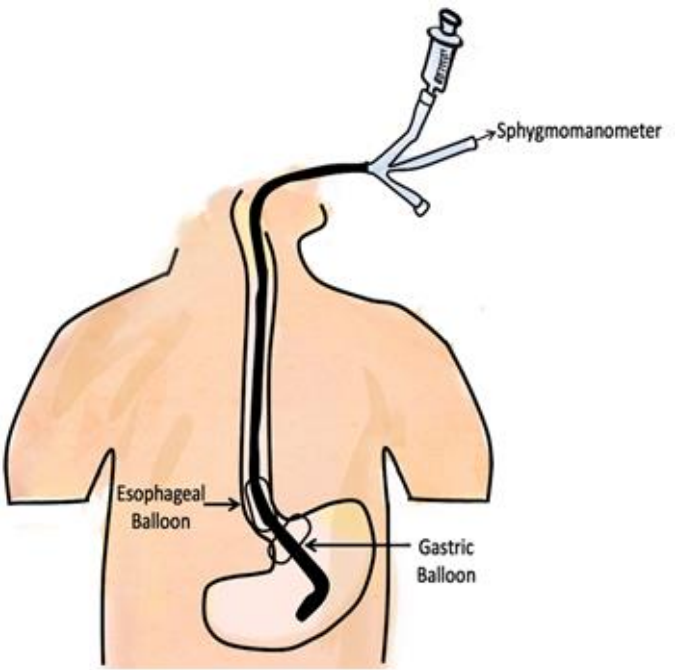


Figure 2. Sengstaken-Blakemore tube placement in a patient.

References

Chojkier M, Conn HO. Esophageal tamponade in the treatment of bleeding varices. A decadel progress report. *Dig. Dis. Sci.* 1980;25(4):267–272.

Hunt PS, Korman MG, Hansky J, Parkin WG. An 8-year prospective experience with balloon tamponade in emergency control of bleeding esophageal varices. *Dig. Dis. Sci.* 1982;27(5):413–416.

Matull W-R, Cross TJS, Yu D, Winslet MC, O'Beirne J. A removable covered self-expanding metal stent for the management of Sengstaken-Blakemore tube-induced esophageal tear and variceal hemorrhage. *Gastrointest. Endosc.* 2008;68(4):767–768; discussion 768.

Paquet KJ, Feussner H. Endoscopic sclerosis and esophageal balloon tamponade in acute hemorrhage from esophagogastric varices: a prospective controlled randomized trial. *Hepatology*. 1985;5(4):580–583.

Vlavianos P, Gimson AE, Westaby D, Williams R. Balloon tamponade in variceal bleeding: use and misuse. *BMJ*. 1989;298(6681):1158.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 8

ECMO for the Intensivist

Orlando Debesa, MD[2]*, Rohan Arya, MD[3], and Sri Sujanthy Rajaram, MD, MPH[1]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Medical College of Virginia, Richmond, VA, US
[3]Cooper University Hospital, Camden, NJ, US

Introduction

Extracorporeal Membrane Oxygenation (ECMO) is a form of external artificial circulation used to augment oxygenation in patients with cardiac and respiratory failure. The two most commonly used ECMO modes are: veno-arterial ECMO (VA-ECMO) or veno-venous ECMO (VV-ECMO).

Over the past few decades, ECMO has not demonstrated benefit mainly because of the lag-time interval between the onset of disease and onset of extracorporeal support, the extended time on mechanical ventilation before initiating ECMO, bleeding complications, and the oxygenation/ventilation capacity-limits of the ECMO circuits used at the time.

In recent years, in lieu of the encouraging results obtained from the CAESAR trial and the H1N1 pandemic, immense progress has been made in constructing ECMO circuits that are more biocompatible, smaller, and capable of lasting longer.

Both VA- and VV-ECMO require blood to be drained from the patient's deoxygenated venous system via gravity into a "bladder." It is then actively pumped through the oxygenator (a synthetic membrane lung) and returned to the patient either through the arterial or venous system. This return cannula is what defines the ECMO mode as either veno-arterial or veno-venous.

Manipulating the oxygen delivery and oxygen consumption ratio, one can determine the adequacy of tissue perfusion by measuring the mixed venous oxygen saturation – the

* Email: odebesa@hotmail.com

percentile amount of oxyhemoglobin that is “left over” after meeting the tissue oxygen demands.

One can estimate individualized ECMO flows by using the following equation:

Flow = 60-100 mL / kg / min

It is important to use a venous drainage cannula with the largest lumen and shortest length to improve its gravitational effects. As long as preload is adequate, the limiting factor in determining adequate flow is cannula resistance, which is determined using Poiseuille law. It is worth noting that catheters of the same size might have varying inner diameters because of the differences in wall thickness. To overcome this, an “M” number is assigned to the catheter, which represents its resistive factor and can be used to approximate its expected flow.

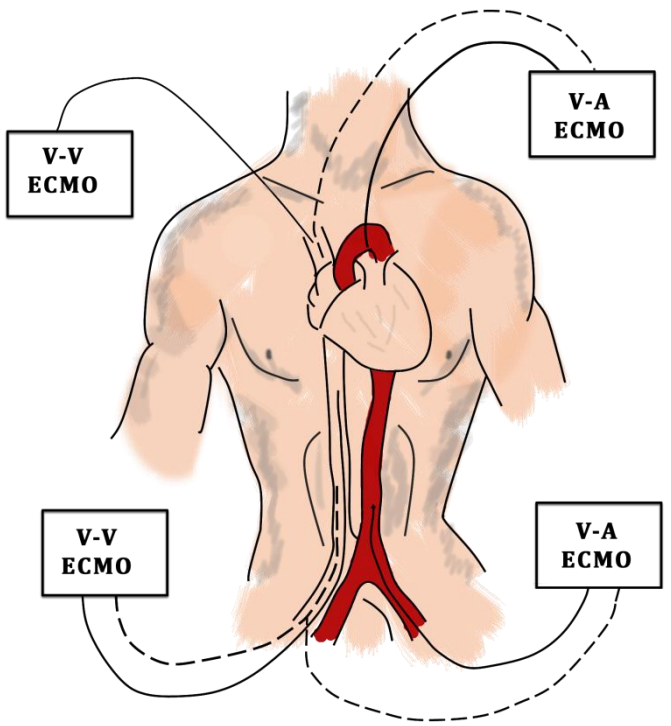


Figure 1. Various cannulation sites for ECMO.

VA-ECMO

VA-ECMO provides both cardiac and respiratory support. Variations in cannulation sites exist but the end result is removal of blood from the right atrium (drainage cannula), the exchange of gases in the oxygenator, and the motor propulsion of the blood back into the aorta (return cannula).

The drainage cannula is typically placed either centrally in the OR or in the right jugular vein or femoral vein. It should ideally be positioned at or near the right atrium. The return cannula is placed centrally or in the right carotid artery or femoral artery. When the femoral artery is used as the return, cannulation should occur up to the level of the descending aorta. One must be aware of the tendency for increased left ventricular afterload, worsening left ventricular function leading to ventricular distention, cardiac injury, and pulmonary edema that might occur with retrograde blood flow. In addition, as the native cardiac function improves in the setting of ill lungs, the retrograde ECMO blood flow competes with the native heart causing compromise of oxygen delivery to the aortic arch, its branches and the coronary arteries.

This situation should be suspected when hypoxemia exists with right radial arterial blood gas analysis or if a disparity in pulse oximetry between the right and left hands exist. Distal limb perfusion may be compromised with femoral placement of the arterial cannula, necessitating the need for a distal leg perfusion cannula.

VV-ECMO

VV-ECMO only provides respiratory support. It is an ideal mode for respiratory failure by allowing ventilatory support to be reduced and further quelling the ill-effects of ventilator-induced lung injury.

The return (oxygenated) ECMO blood mixes with systemic hypoxic blood in a 3:1 fashion rendering the arterial saturation to be in the 80% range. There are variations in cannulation sites but the central premise is the same: venous drainage, the exchange of gases in the oxygenator and the motor propulsion of blood back into the right atrium. In VV-ECMO, pulsatile flow from the native heart is maintained with minimal hemodynamic effects. Because cardiac output is determined by the patient, pulsatile flow is preserved to the kidneys and other end-organs. In addition, there is low risk of embolic phenomena or reduced extremity flow.

The drainage cannula is often placed centrally, or in the right jugular vein or femoral vein. When the femoral vein is used as drainage, the cannula should ideally be positioned in the IVC at the level of the diaphragm. If the right internal jugular vein is used, the cannula should be placed within the SVC. The separation of the drainage and return cannulas is important to prevent recirculation. The most common VV-ECMO management problem arises from the conflicting goals of maintaining adequate oxygenation (which may require a high flow rate) and a low CVP (which is beneficial for the lungs but may cause limitation of ECMO flow). Hence, the goal is to maintain adequate patient oxygenation at the lowest CVP possible. In practice, an arterial PO_2 of 50-55 mmHg or an oxygen saturation of 85-90% is acceptable. Pushing fluid to maintain a high ECMO flow rate and an oxygen saturation of more than 90% may ultimately result in severe fluid overload. If adequate oxygenation cannot be maintained at a low or normal CVP, a second access line should be inserted.

When native lung function begins to return, an increase in arterial saturation above mixed venous oxygen saturation will be evident, and VV flow rate can be reduced to maintain arterial saturations ~ 90%. Once VV-ECMO flow is decreased to approximately 2 L/min, the patient is ready for a trial off ECMO.

General Contraindications to ECMO

- Non-recoverable cardiac disease
- Non-recoverable pulmonary disease
- Non-recoverable neurological disease
- Chronic severe pulmonary hypertension
- Active malignancy
- Immunosuppression
- Weight greater than 140 kilograms
- Advanced liver disease
- Unwitnessed cardiac arrest or CPR > 60 minutes prior to commencement of ECMO
- Contraindication to anticoagulation
- Intracranial hemorrhage
- Refusal to receive blood products

Specific Indications and Contraindications

	Indications	**Contraindications**
VA-ECMO	• Reversible cardiac failure/cardiogenic shock	• Aortic Dissection • Severe Aortic Regurgitation
VV-ECMO	• Reversible respiratory failure • Murray score >3 • Uncompensated hypercarbia with pH <7.2	• >10 days on mechanical ventilation • >7 days on high-pressure mechanical ventilation

ECMO Circuit Components

Pumps

There are two types of pumps - roller and centrifugal. The pump is used to direct blood forward to a dedicated outlet. Patients on VV-ECMO do not require a pump as their heart is working.

Gas Exchange Device (Oxygenator)

The gas exchange device is designed to add oxygen and remove CO_2. The device must be adequate in size to provide for the anticipated metabolic requirements of the patient. Its surface area and blood path mixing determines its maximum oxygen capacity. Rated flow is the standard that is used to compare the different gas exchange devices; it is defined as the flow rate at which venous blood, with a saturation of 75% and a hemoglobin of 12 g/dL, will

exit the gas exchange device with a saturation of 95%. A gas exchange device should be chosen that is capable of total support.

Sweep Gas

Sweep is the rate of gas flow to the oxygenator. For most applications, sweep gas will be 100% oxygen. The rate of sweep gas determines CO_2 removal from the blood through the oxygenator. Increasing the sweep gas flow rate will increase the CO_2 clearance, but will not affect oxygenation. Decreasing the sweep gas flow rate will increase pCO_2, but will usually not affect oxygenation. Water vapor can condense within the gas compartment of the membrane lung and may be cleared by intermittently increasing sweep gas flow to a higher flow. If there is excessive water that has not been purged, the pCO_2 may rise.

Anticoagulation

Clot formation is an unfortunate consequence of extracorporeal circuits. The non-biological surfaces encountered by blood propagate this process. For this reason, the circuitry must be regularly checked for clot formation, which may develop most commonly within the pump head or on the inflow side of the oxygenator. If the oxygenator is adversely affected, it may warrant an oxygenator change-out. Heparin is usually the anticoagulant of choice and a loading dose is usually administered before cannulation occurs (5000-10,000 units). Therapeutic heparin levels are measured using ACT (usually between 150-180 seconds). Heparin resistance has been reported and is usually due to ATIII deficiency and is treated using fresh frozen plasma. Flow below 2 L/min for prolonged periods of time must be avoided.

References

Bartlett, RH, Gattinoni L. Current status of extracorporeal life support (ECMO) for cardiopulmonary failure. *Minerva Anestesiologia*, July 2010.

Cordell-Smith JA, Roberts N, Peek GJ, Firmin RK. Traumatic lung injury treated by extracorporeal membrane oxygention (ECMO). *Injury* 2006; 37:29-32.

Maggio P, Hemmila M, Haft J, Bartlett R. Extracorporeal life support for massive pulmonary embolism. *J. Trauma* 2007; 62: 570-576.

Mikkelsen ME, Woo YJ, Sager JS, et al. Outcomes using extracorporeal life support for adult respiratory failure due to status asthmaticus. *ASAIO J.* 2009; 55:47-52.

Morris AH, Wallace CJ, Menlove RL, et al. Randomized clinical trial of pressure-controlled inverse ratio ventilation and extracorporeal CO2 removal for adult respiratory distress syndrome. *Am. J. Respir Crit Care Med*. 1994; 149: 295-305.

Peek, M, Mugford M, Tiruvoipati R, et al. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult

respiratory failure (CESAR): a multicentre randomised controlled trial. *The Lancet* 2009; 374:1351-1363.

Rich PB, Awad SS, Crotti S, Hirshl RB, Bartlett RH: A prospective comparison of atrio-femoral and femoro-atrial flow in adult veno-venous extracorporeal life support. *J. thorac Cardiovasc Surg.* 116: 628-32, 1998.

Sidebotham D, McGeorge, A. Extracorporeal membrane Oxygenation for treating severe cardiac and respiratory failure in Adults. *Journal of Cardiothoracic and Vascular Anesthesia*, Vol 24, No 1, February 2010: 164-172.

Wang, D, Zhou X, Liu X, Zwischenberger JB. Wang-Zwische double lumen cannula-toward a percutaneous and ambulatory paracorporeal artifical lung. *ASAIO J.* Nov-Dec; 54(6): 606-11, 2008.

Zapoli WM, Snider MT, Hill JD, et al. Extracorporeal membrane oxygenation in severe acute respiratory failure. A randomized prospective study. *JAMA* 1979; 242:2193-2196.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 9

Feeding Tubes and Percutaneous Tubes

Shipali Pulimamidi, MD[1], Carol Choe[*], MD[1]
and Sri Sujanthy Rajaram, MD, MPH[2]
[1]Cooper Medical School of Rowan University, Camden, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Nutritional support in the critically ill is an integral component of care and affects the overall morbidity and mortality. Enteral nutrition is a more physiologically ideal approach to provide nutrition for this subset of patients when compared to parenteral nutrition. Based on the duration of need for invasive enteral feeding there are different feeding tube options available. For short-term feeding, such as less than one month, nasogastric (NG) or orogastric (OG) tubes are a good option. However, if the clinical situation requires a more prolonged feeding option then percutaneous gastrostomy tubes (PEG), jejunostomy tubes (PEJ) or gastrojejunostomy tubes (PEG-J) are available.

Indications

- Nasal or oral gastric and nasal or oral jejunal tubes are indicated for temporary situations in the ICU for feeding as well as oral medication administration.
- PEG, PEJ and PEG-J tubes are indicated if the patient requires enteral feeding for more than a month in situations such as malignancy, stroke, dementia, disabling neurological conditions, facial trauma and prolonged ventilation assistance.

[*] Email: dr.carolchoe@gmail.com.

- Large bore NG tubes are used for gastric lavage and suctioning of gastric contents. Some post operative patients, Ileus and Intestinal obstruction NG tube is utilized for continuous suctioning.

Figure 1. Dobhoff nasogastric feeding tube.

Preparation

PEG tube placement

- Placement can be done at bedside, in the endoscopy suite, or in the operating room.
- Obtain informed consent (from patient or family)
- Visualize the patient's abdomen. If there is any presence of gastric outlet obstruction, a jejunostomy feeding tube may be required.
- Preoperative antibiotic prophylaxis may be given – usually a first generation cephalosporin; surgeon dependent.
- Place the patient on a cardiopulmonary monitor for the procedure
- Determine which sedative or anesthetic to administer
- Position the patient appropriately – usually supine with head of bed at 30°.

Procedure

NG tube placement or OG tube placement: Flex the neck and insert the tip of the tube through nose or mouth after lubrication. Placement of a large bore OG or NG tube can be

checkd by listening for air inflation on the abdomen by palcing a stethoscope. Definite placement of the feeding tubes must be confirmed by chest X-ray.

- Typically, an 8- to 12-Fr NG tube is lubricated and passed through the nare into the stomach with the patient's head flexed; the patient ingests sips of water to assist in passing the tube into the stomach.

PEG tube: The most common sizes for adult patients range from 16 to 24 Fr. Most tubes are made of silicone, although some are constructed of polyurethane.

- Prep and drape the patient’s abdomen using sterile technique
- Adequately sedate the patient and pass the endoscope via the mouth into the stomach.
- Using trans-illuminationvisualize and insufflate the stomach.
- Identify the insertion site on the stomach, approximately 2cm from costal margin. Mark the area and inject lidocaine.
- A 1cm incision is made with a blade and a 14-18 gauge needle is passed through the incision into the stomach while viewing via endoscope.
- A braided suture is passed through the needle and encircled by a snare passed through the endoscope. Once the “rope” is securely entrapped, the needle is removed and the entire endoscope with snare and attached rope is withdrawn through the mouth
- The lubricated feeding tube is then attached to the rope. The assistant then withdraws the rope from the stomach wall, and the tube is carefully guided through the patient's mouth into the stomach and is pulled into position.
- Once the feeding tube has been drawn through the skin to approximately 4 cm, the endoscope is reinserted into the stomach to ensure proper seating of the feeding tube.
- A skin disc is placed to help hold the tube in position against the abdomen.

Complications

Nasoenteric tube complications:

- Arrhythmia
- Empyema
- Gastric perforation
- Myocardial infarction
- Otitis media
- Pulmonary intubation
- Tube feeding into pulmonary tree
- Clogging
- Epistaxis
- GI bleeding
- Nasal mucosal ulceration
- Peumothorax

- Pyriform sinus perforation
- Tracheobronchial trauma
- Tube dislodgement
- Duodenal perforation
- Esophageal perforation
- Knotted tubes
- Nasal trauma
- Pulmonary aspiration
- Trachoesophageal fistula
- Tube obstruction
- Reflux esophagitis
- Ulceration or stricture.

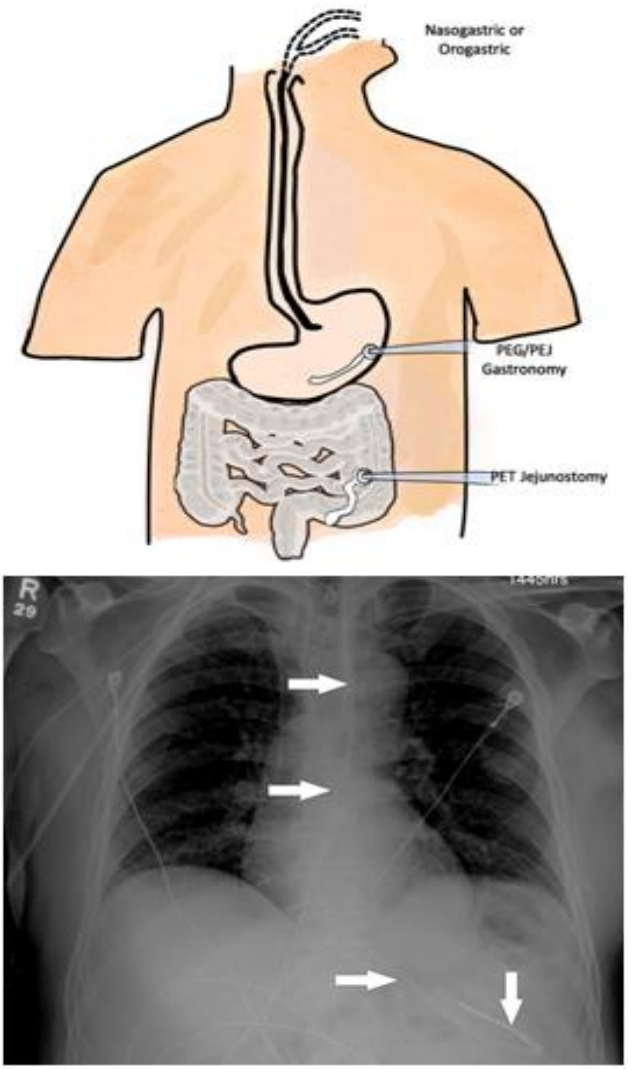


Figure 2. Sites for enteral feeding tube placement. CXR demonstrating placement of dobhoff feeding tube.

Contraindications

Relative contraindications for PEG placement include:

- Presence of gastric varices
- Severe obesity
- Major gastric resection
- Significant disease of the gastric or abdominal wall
- Gastric outlet obstruction
- Oral or upper airway tumor
- Ascites
- Coagulopathy.

Absolute contraindications include:

- Inability to trans-illuminate the anterior abdominal wall
- Ineffective digital intrusion of the abdominal wall to locate a safe gastric access site.

References

Anon. American Gastroenterological Association Medical Position Statement: guidelines for the use of enteral nutrition. *Gastroenterology*. 1995;108(4):1280–1281

Caulfield KA, Page CP, Pestana C. Technique for intraduodenal placement of transnasal enteral feeding catheters. *Nutr Clin Pract*. 1991;6(1):23–26.

Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*. 9th ed. Philadelphia: Saunders; 2010.

Heyland DK, McClave SA. Chapter 11. Nutrition in the Critically Ill. In: Hall JB, Schmidt GA, Wood LH. eds. *Principles of Critical Care, 3e.* New York, NY: McGraw-Hill; 2005. http://accessmedicine.mhmedical.com. proxy.lib. umich.edu/content.aspx?bookid=361& Sectionid=39866377. Accessed May 22, 2014.

Kahrilas PJ, Clouse RE, Hogan WJ. American Gastroenterological Association technical review on the clinical use of esophageal manometry. *Gastroenterology*. 1994;107(6):1865–1884.

Safadi BY, Marks JM, Ponsky JL. Percutaneous endoscopic gastrostomy. *Gastrointest. Endosc. Clin. N. Am.* 1998;8(3):551–568.

Townsend CM, Evers BM, eds. Atlas of General Surgical Techniques. 1st ed. Philadelphia, PA: Saunders; 2010: 253-260.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 10

FoCUS Echo in the Critical Care Setting

Ben Goodgame, MD[1], David Schrift, MD[2], Patricia E. Walley, Vivek Punjabi, MD[3] and Sri Sujanthy Rajaram, MD[4]

[1]Lakes District Health Board, Rotorua Hospital,Rotorua, New Zealand
[2]University of South Carolina School of Medicine, Columbia, SC, US
[3]Cardiac Echo Laboratory, St Paul's Hospital,
University of British Columbia, Vancouver, Canada
[4]Mercy Hospital, Springfield, MO, US
[5]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Key Points

- Having a systematic, consistent approach to obtain the multiple views of the FoCUS (Focused Cardiac Ultrasound) exam is necessary to ensure precision and accuracy.
- FoCUS Echo should be performed to answer a specific clinical question (e.g. Why is my patient hypotensive?). The major advantage of FoCUS Echo is that the provider is performing the study; thus the provider can combine a patient's clinical features with the FoCUS exam to improve diagnostic accuracy and therapeutic management.
- The FoCUS exam can be combined with other focused ultrasonographic exams (lung ultrasound, vascular ultrasound etc.) to enhance the accuracy of the FoCUS exam.
- FoCUS Echo is easily repeated and should be employed if the patient's status changes and/or an intervention is performed.
- When the FoCUS Echo exam is insufficient or when additional abnormalities are appreciated, order a comprehensive cardiac echocardiogram.

Introduction

The use of FoCUS Echo in the critical care setting has become an invaluable tool as an adjunct to, or component of, "goal directed" therapies. FoCUS Echo is a time-sensitive examination performed on a symptomatic patient. Compared to a conventional comprehensive cardiac Echo exam, FoCUS Echo has a more limited scope that specifically addresses the clinical questions of the physician. These clinical questions are numerous. Examples include:

- Why is my patient hypotensive?
- Why is my patient dyspneic?
- Why is my coding patient in PEA?
- Will my patient benefit from fluids?

When integrating the FoCUS exam into the diagnostic and therapeutic management of one's patient, it is important to always combine the FoCUS exam with existing clinical information (history, exam findings, laboratory data, imaging and hemodynamics, etc.). For example, a patient with a pericardial effusion with tamponade physiology does not need a pericardiocentesis if they are hemodynamically stable and asymptomatic.

It is important to remember that FoCUS Echo is different than the comprehensive cardiac echocardiograms performed by fully trained and experienced sonographers and physicians. Comprehensive cardiac Echo exams are designed to assess additional aspects of cardiac structure and function, they require more time, additional views, additional tools (IV contrast, additional maneuvers, specific drug administration, etc), and a greater understanding of the many abnormal findings encountered in a wide patient population. In conducting a FoCUS Echo exam you will be imaging the most technically difficult patients in the most challenging situations (lung interference, mechanically ventilated, positioning limitations, and the urgency to acquire the information for immediate treatment). It is vital to recognize when you fail to acquire necessary images at which point order a comprehensive echocardiogram.

Indications

- To answer a specific clinical question
- Specific goals are to evaluate:
  - Left ventricular (LV) size and function
  - Right ventricular (RV) size and function
  - Pericardial effusion and potential signs of hemodynamic compromise
  - Size and distensibility of the inferior vena cava (IVC) for evaluation of volume status

Contraindications

None

Preparation

Machine placement

- Place the machine on the side of the bed that allows you to image the patient with your preferred hand.
- Take care to position the patient, machine, and height of the bed for your comfort in order to decrease the risk of musculoskeletal discomfort and possible injury. You should have the machine in front of you, so you are facing the patient and the machine.

Patient position

- You should place the patient into the left lateral position if you cannot obtain the required images with the patient in the supine position.

Transducer type

- The most frequently used transducer is the 2.5 Mhz phased array probe for adequate penetration to image cardiac structures (Figure 1). Transducers with a higher frequency have better resolution at the expense of decreased tissue penetration. The "footprint" or size of the 2.5 Mhz transducer head is designed to be small enough to fit between the intercostal spaces (ICS).

Initial settings

- The initial depth is increased to 20-24 cm to identify pericardial and pleural effusions.
- The depth should then be decreased so that the entire structure of interest occupies the screen.
- Gain should be adjusted to see endocardial borders clearly. Be careful to not "overgain" (too bright, washes out image) or "undergain" (overly-dark echo images) **(Figure 2a, 2b)**

Figure 1. 2.5 Mhz phased array probe.

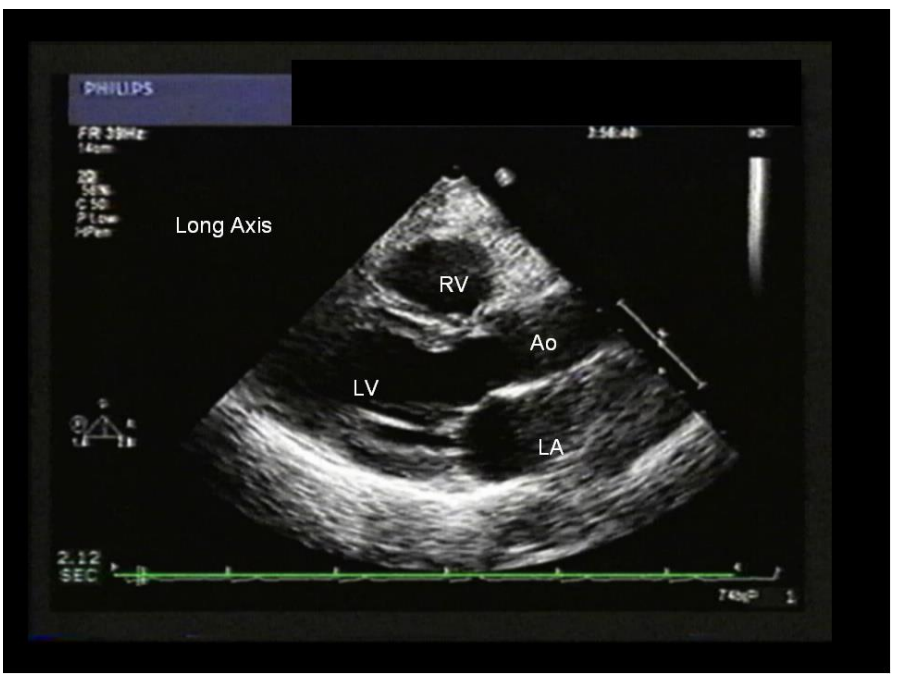


Figure 2.

Procedure

FoCUS Echo is comprised of 4 main views. Use a "clockwise positioning approach," starting from the parasternal views to the apical view and finally the subcostal approach. If you are unable to obtain an adequate image in one view, move on to the next view when necessary. You may need to go directly to the subcostal view, which is frequently the easiest to obtain and provides the most information, including the inferior vena cava (IVC) for assessment of volume status.

Parasternal Long-Axis View (PLAX) (Figure 3)

Goal: Assessment of LV size and function, RV size, pericardial effusion, and sometimes a left pleural effusion.

1. Using the cardiac preset, start at the second or third intercostal space (ICS), as close to the sternum as possible, with the transducer marker directed at approximately 11 o'clock (towards the right shoulder).
2. Move the transducer up or down an ICS to search for the best view, when necessary. The structures should appear horizontal on the screen. Keep the image in the middle of the screen by adjusting transducer position.
3. Increase the depth to 20-24 cm to image the presence of a possible pericardial effusion or pleural effusion.
4. Decrease the depth to approximately 14-16 cm, depending on the size of the heart to fill the screen with cardiac structure.

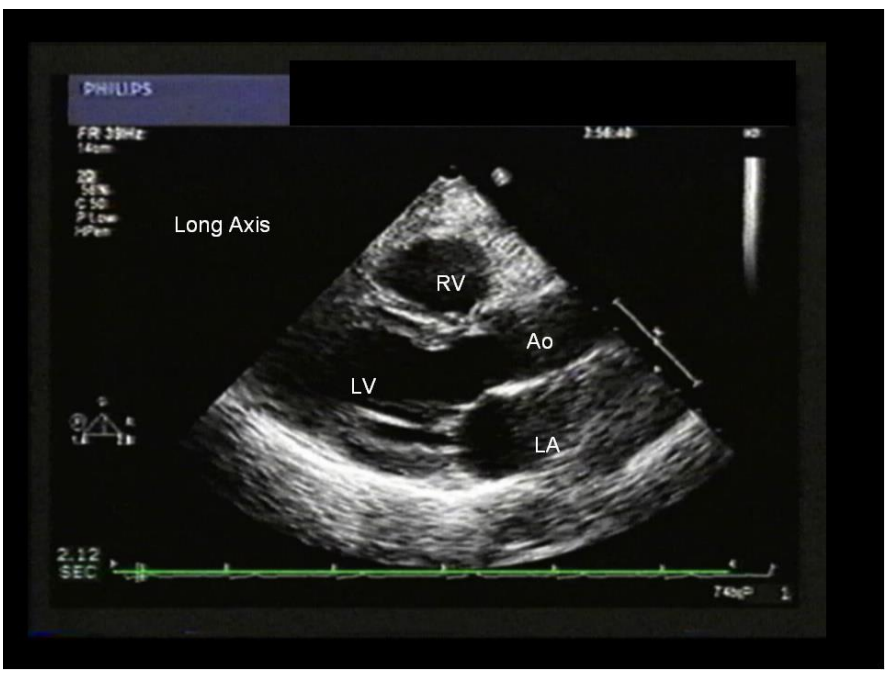


Figure 3. Parasternal long axis. RV, right ventricle; LV, left ventricle; Ao, aorta; LA, left atrium.

Parasternal Short-Axis View (PSAX) (Figure 4)

Goal: Assessment of LV size and function, including the presence of abnormal septal motion and the "D" sign (RV pressure overload), pericardial effusion, and the presence of regional wall motion abnormalities. This is also known as the transverse view.

1. Using the cardiac preset and from the PLAX position, rotate the transducer clockwise until the transducer marker is at approximately 2 o'clock (towards the left shoulder).
2. Base of the LV at the level of the mitral valve:
   a. Angle or tilt the transducer (soundwave beam) slightly laterally and inferiorly to image the LV with both mitral leaflets in view. The mitral valve apparatus should appear "fish-mouthed."
3. Mid-LV
   a. Continue angling the transducer (soundwave beam) slightly inferiorly and laterally until you see the tips of the papillary muscles within the LV.
   b. If the patient is volume depleted/hypovolemic, the LV may appear small and you may see the mitral leaflets at all levels.
   c. The LV should appear round. If it appears "egg-shaped" or oblong, you need to slide the transducer up an ICS.

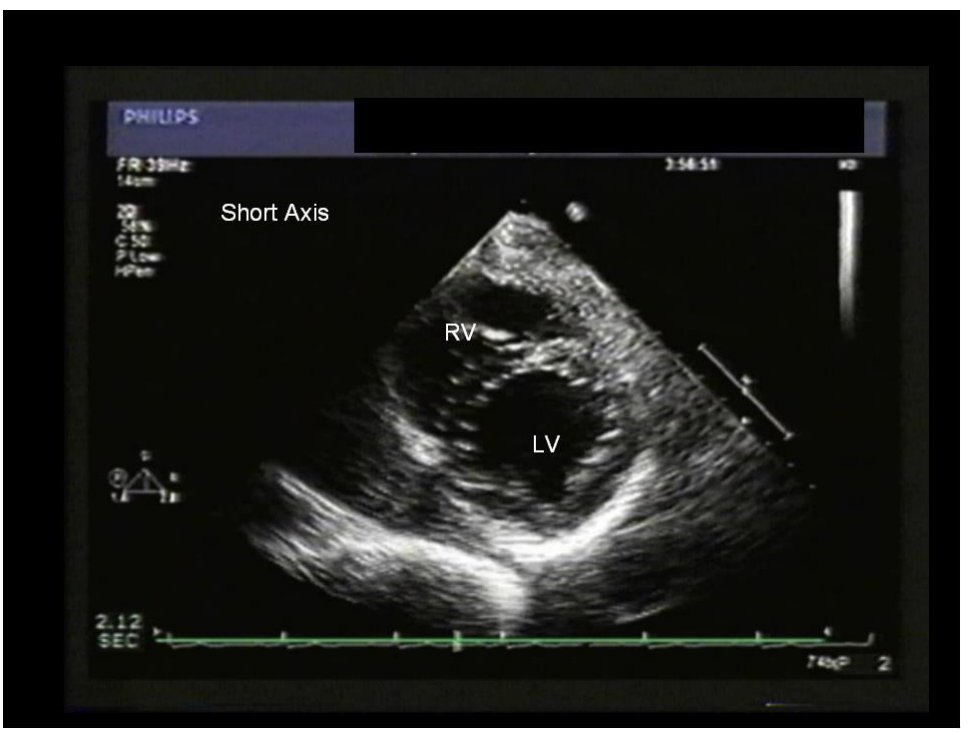


Figure 4. Parasternal short axis with view of the mitral valve. RV, right ventricle; LV, left ventricle.

Apical 4-Chamber View (A4) (Figure 5)

Goal: Assessment of LV size and function, RV size and function, and pericardium; assessment of RV/LV ratio; assessment of pericardial effusion and evidence of hemodynamic compromise, and sometimes, left pleural effusion.

1) Using the cardiac preset, rotate the transducer clockwise with the transducer marker at approximately 3 o'clock.
2) It is helpful to feel for the PMI. Place the transducer slightly lateral to the PMI and angle the transducer or sound wave beam up through the apex of the heart to the right shoulder.
3) On the screen, the right and left ventricles will be at the top of the screen with the atria below.
4) The LV is shown on the upper right side of the screen and the RV is shown on the upper left side of the screen.
5) Ensure the atria and ventricles are in a vertical axis and are not tilted to the right or left.

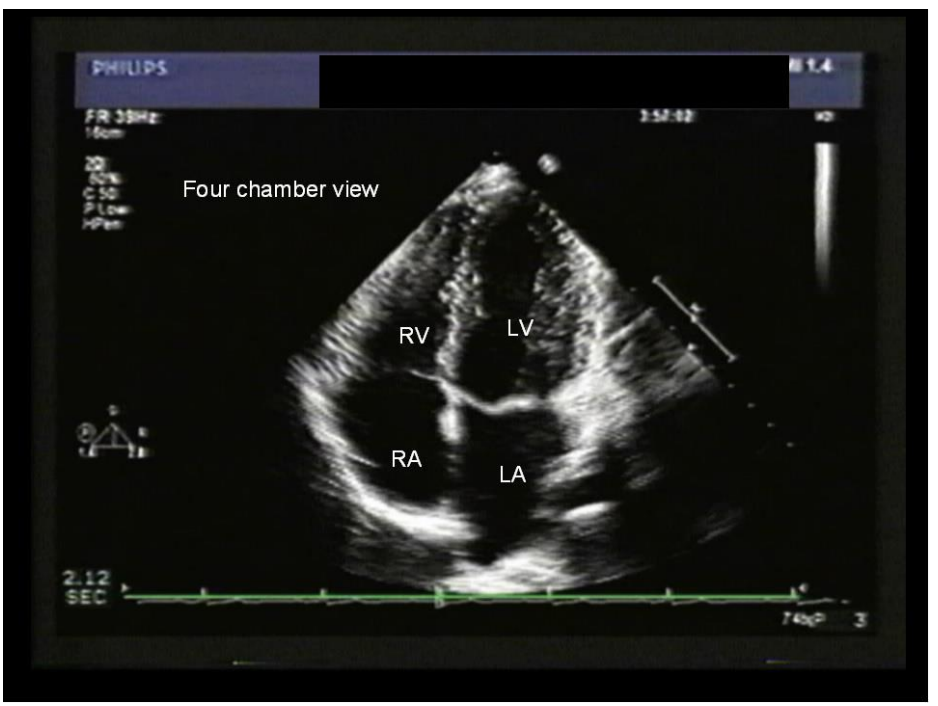


Figure 5. Apical 4-chamber view. RV, right ventricle; LV, left ventricle; RA, right atrium; LA, left atrium.

Subcostal View (Figure 6)

Goal: Measurement of IVC collapse to assess volume status; assessment of all four chambers with LV and RV size and function; assessment of pericardial effusion and evidence of hemodynamic compromise; evaluation of presence of a right pleural effusion.

1) Transducer at the subxyphoid position and angled slightly medially, pointing the soundwave beam to the patient's left side. The transducer should be almost flat against the abdominal wall.
2) For imaging of the IVC, angle or tilt the transducer and sound wave beam medially to view the right atrium (RA). Slightly rotate the transducer counter-clockwise. Tilt the transducer and soundwave beam slightly inferiorly to "open up" the image of the IVC in the long axis plane (Figure 7).
3) The IVC should be measured about 2 cm from the RA/IVC junction or just distal to the confluence of the hepatic vein and IVC.

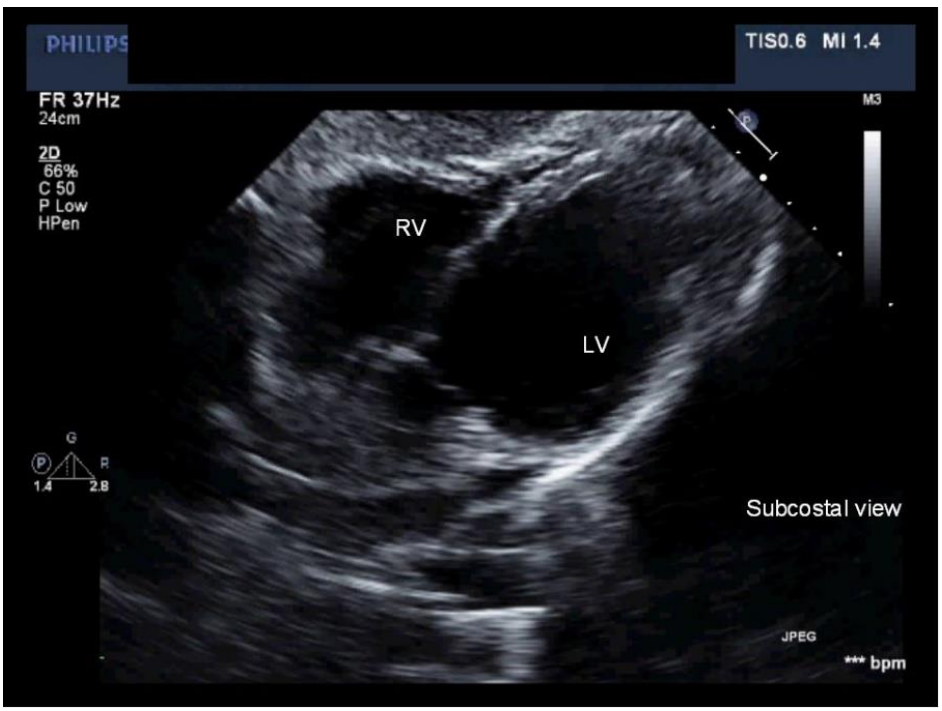


Figure 6. Subcostal view. RV, right ventricle; LV, left ventricle.

Interpretation

Full interpretation, which involves linking the FoCUS Echo findings to hemodynamic and other clinical information, is beyond the scope of this chapter. A key caveat is that you **must** see endocardial borders to accurately assess RV and LV function.

- Questions to be asked:
  - Left ventricle: Dilated or not?
  - Left ventricular function: "Significantly" (moderately or severely) impaired?
  - Right ventricle: Dilated or not?
  - Right ventricular function: "Significantly" impaired?

- Is there evidence of hypovolemia? (small/empty left ventricle, IVC collapsing > 50%)
- Pericardium: Presence or absence of pericardial effusion

- Additional findings should be noted:
  - Are valvular abnormalities, wall motion abnormalities, etc, evident? This should trigger a comprehensive Echo exam.
  - Pleura: Is there evidence of a pleural effusion?

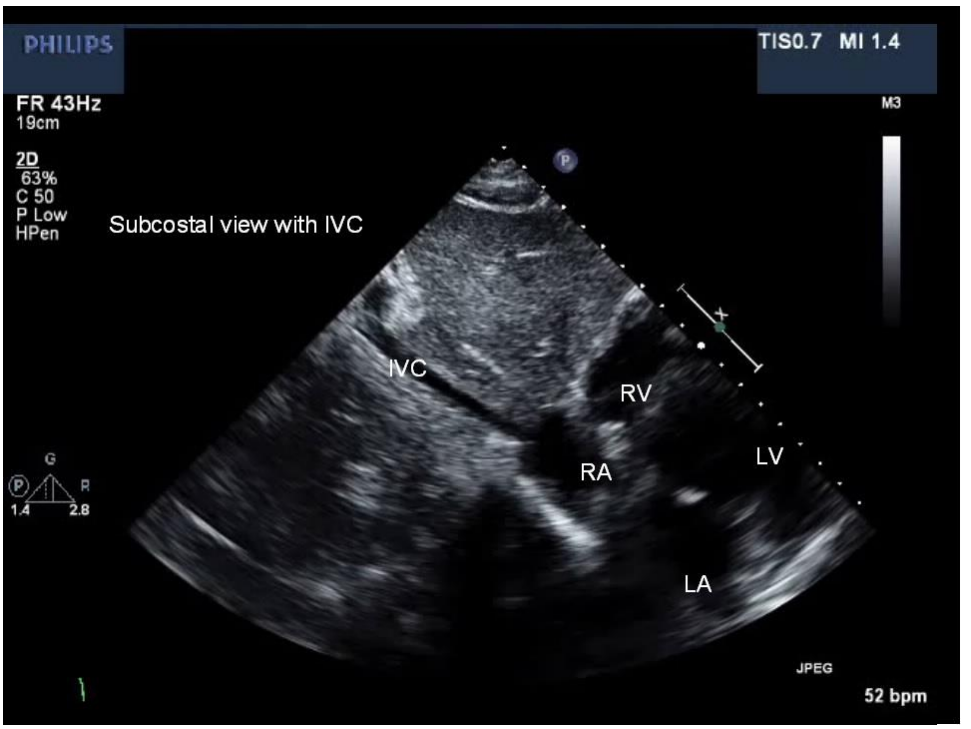


Figure 7. Subcostal view with visualization of the IVC merging with the right atrium. IVC, inferior vena cava; RA, right atrium; RV, right ventricle; LA, left atrium; LV left ventricle.

Shock States

- **Distributive shock:** In less severe or in earlier stages of shock, the Echo may show a hyperdynamic LV and a smaller-to-normal size IVC with > 50% collapsibility as septic shock is often initially associated with a degree of hypovolemia.
  - Additionally, there is a separate cohort of patients with distributive shock related to severe sepsis who also have impaired LV function as part of their pathologic state.
- **Hypovolemic shock:** Echo shows small ventricles, sometimes "near cavity obliteration" due to a small and hyperdynamic LV. A small IVC with wide respiratory variation, which collapses > 50% on inspiration in a spontaneously breathing patient is also noted.

- **Obstructive shock:** If due to tamponade (Figure 8a) (evidence of hemodynamic compromise), Echo will *usually* show a significant pericardial effusion (usually encompassing the heart) with right atrial (RA) wall late diastolic collapse and right ventricular (RV) wall early diastolic collapse.
  - The IVC is dilated or plethoric.
  - In regards to evaluating a patient with suspicion of tamponade, what you are looking for on FoCUS Echo is *tamponade physiology*. However, tamponade still remains a clinical diagnosis.
  - If due to acute pulmonary embolism (Figure 8b), Echo will show a dilated RV and an RV/LV diameter ratio >1:1 and/or a D-shaped septum (Figure 9).
  - In the hemodynamically unstable patient, the FoCUS exam is extremely accurate in ruling in or out a PE. The FoCUS exam is less accurate in a hemodynamically stable patient.
  - The FoCUS exam can be combined with a focused vascular exam for the evaluation of a DVT to further increase its accuracy
- **Cardiogenic shock:** Echo will show a dilated LV with significantly decreased LV function, possibly decreased RV function and a dilated IVC
  - Lung ultrasound will show B-lines (narrow, vertical, hyperechoic lines arising from the pleural edge and extending down the length of the lung field) in all lung fields and can improve your confidence in the diagnosis.

References

Feissel M, Michard F, Faller J-P, Teboul J-L. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med*. 2004;30(9):1834–1837.

Himelman RB, Kircher B, Rockey DC, Schiller NB. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. *J. Am. Coll. Cardiol.* 1988;12(6):1470–1477.

Jardin F, Dubourg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. *Chest*. 1997;111(1):209–217.

Labovitz AJ, Noble VE, Bierig M, et al. Focused cardiac ultrasound in the emergent setting: a consensus statement of the American Society of Echocardiography and American College of Emergency Physicians. *J Am Soc Echocardiogr*. 2010;23(12):1225–1230.

Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005;18(12):1440–1463.

Levitov A, Marik PE. Echocardiographic assessment of preload responsiveness in critically ill patients. *Cardiol Res Pract*. 2012;2012:819696.

Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N. Engl. J. Med.* 2001;345(19):1368–1377.

Sagristá Sauleda J. [Clinical decision making based on cardiac diagnostic imaging techniques (I). Diagnosis and therapeutic management of patients with cardiac tamponade and constrictive pericarditis]. *Rev Esp Cardiol*. 2003;56(2):195–205.

Via G, Hussain A, Wells M, et al. International evidence-based recommendations for focused cardiac ultrasound. *J Am Soc Echocardiogr*. 2014;27(7):683.e1–683.e33.

Vincent JL, Rhodes A, Perel A, et al. Clinical review: Update on hemodynamic monitoring--a consensus of 16. *Crit Care*. 2011;15(4):229.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 11

Intraosseous Access

Jonathan D. Trager, DO[1,2]
[1]Departments of Emergency Medicine and Critical Care,
St. Luke's University Health Network, Bethlehem, PA, US and
[2]Department of Emergency Medicine,
Temple University School of Medicine, Philadelphia, PA, US

Introduction

- Placement of an intraosseous needle is an essential skill.
- The needle is placed into the marrow canal of a long bone (femur, tibia) or flat bone (sternum, iliac crest).
- Multiple sites (the iliac crest, femur, proximal and distal ends of the tibia, radius, clavicle, and calcaneus) may be used.
- Most medication that can be administered intravenously can be given via the intraosseous route.
- There are many types of intraosseous needles available (therefore, you will need to become familiar with the device at your institution).

Indication

- Immediate resuscitation
- Cardiac arrest in infants and young children
- IV access cannot be achieved quickly or reliably
- Adult patients in whom attempts at peripheral or central venous access have been unsuccessful

Contraindication

- Osteoporosis
- Osteogenesis imperfecta
- Fractured bone
- Recent IO puncture sites placed in the same bone
- Overlying areas of cellulitis, infection, or burns
- Patients with right-to-left intracardiac shunts (may be at higher risk for fat or bone marrow embolization)

Preparation

Equipment:

- IO device
- Towels
- 4X4s
- Tape
- 10 cc syringe
- IV tubing
- IV extension kit (may come with IO kit)
- Lidocaine (2% preservative-free lidocaine without epinephrine)

Procedure

- Site of placement depends on age and size of patient (Child: proximal tibia, distal tibia, distal femur; Adult: distal tibia, humeral head, proximal tibia, distal femur, sternum)
- Cleanse insertion site similar to a peripheral IV site (cleansing agent required by specific protocol)
- When inserting the needle, Lidocaine can be used to provide local anesthesia (use institutional protocols)
- Locate the preferred landmark:

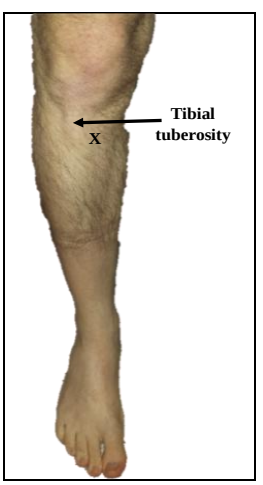
General Procedure

This will vary based on equipment used. Procedure described is for manual insertion. With automatic devices such as EZ-IO, follow the manufacture instructions. The general principles are similar however.

- Find the appropriate landmark.
- Insert the IO needle through the skin and subcutaneous tissue.
- Stabilize the needle with the index finger and thumb.
- Apply constant pressure on the needle with the palm of your hand while applying a twisting motion to advance the needle. (A 10-15° caudal angulation may be used but typically the needle is perpendicular- 90°to the bone). If using an automatic drill device no manual twisting is needed.
- When the needle enters the marrow space you may feel a popping sensation or lack of resistance.
- STOP! The needle should now stand on its own.
- Remove the inner trocar, attach a syringe to the needle, and aspirate bone marrow. Obtaining marrow confirms placement but may not occur.
- If marrow is not aspirated, flush with approximately 10-mL of isotonic sodium chloride solution. There should be no resistance to flow and extravasation should not be evident.
- Observe for tissue swelling- if noted, do not use the needle and remove it safely.

Proximal Tibia

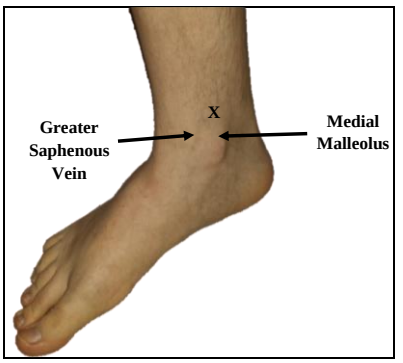
- Identify the tibial tuberosity, just below the knee.
- Locate a consistent flat area of bone 2 cm distal and slightly medial to the tibial tuberosity.
- Support the flexed knee by placing a towel under the calf.
- Observe the calf area for swelling.



Distal Tibia

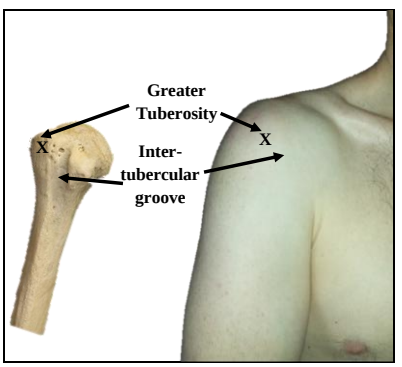
- Palpate the flat portion of distal tibia over the medial surface at the junction of the medial malleolus and tibial shaft, posterior to greater saphenous vein

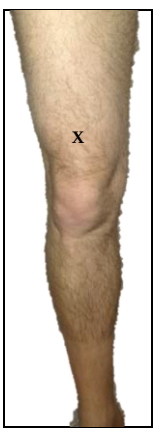
- Slightly abduct and externally rotate the hip to expose the site.
- Angle the needle 10-15° cephalad to minimize the risk of growth plate injury.



Humeral Head

- Position the patient so the shoulder is adducted and internally rotated so that the greater tuberosity is most prominent.
- Palpate the proximal humerus and identify the greater tuberosity.
- Insert the needle at a 90-degree angle directly into the greater tuberosity (the most prominent aspect of the greater tubercle, 1 to 2 cm above the surgical neck and 1-2 cm lateral to the inter-tubercular groove.)





Distal Femur

- Slightly flex and externally rotate the hip, and flex the knee so that the quadriceps are relaxed.
- Insert the needle in the anterior midline, above the lateral epicondyles, 1-3 cm above the femoral plateau.

Post-Procedure Care

NO FLUSH = NO FLOW!

- Once the needle is in place and confirmed to be in proper position by aspiration of marrow and/or easily flushing fluid, the needle should be secured.
- To secure the needle, place a stack of 4X4 dressings on either side of the needle and tape in place.
- Some needles may come with their own securing device which she be used and 4X4s can be used to provide additional support**.**

Complications

- Fracture
- Infection
- Compartment syndrome
- Skin sloughing
- Fat embolism
- Pain with insertion
- Epiphyseal injury
- Fluid extravasation
- Needle obstruction

References

http://emedicine.medscape.com/article/940993-overview Pediatric Intraosseous Access, Accessed March 20, 2015

http://reference.medscape.com/article/80431-overview#a01 Intraosseous Access, Accessed March 20, 2015

Michael W. Day. Intraosseous Devices for Intravascular Access in Adult Trauma Patients. *CriticalCareNurse,* Vol 31, No. 2, APRIL 2011: 76-89

Recommendations for the Use of Intraosseous Vascular Access for Emergent and Nonemergent Situations in Various Health Care Settings: A Consensus Paper; The Consortium on Intraosseous Vascular Access in Healthcare Practice. *CriticalCareNurse,* Vol 30, No. 6, DECEMBER 2010, e1-e7

Roberts and Hedges Clinical Procedures in Emergency Medicine 6th Edition Elsevier Saunders

The Science &Fundamentals of Intraosseous Vascular Access Vidacare Science & Clinical Team December 2013 Vidacare Corporation

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 12

Intra-Abdominal Pressure Monitoring

Sachin Mohan, MD[2] and Sri Sujanthy Rajaram, MD, MPH[1*]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US

Introduction

Intra-abdominal pressure monitoring is used to evaluate for or trend the development of intra-abdominal hypertension (IAH) or abdominal compartment syndrome (ACS).

The monitoring system uses intravesicular pressure as a surrogate for intra-abdominal pressure (IAP). It is affected by multiple factors including the volume of solid organs and hollow viscera; presence of ascites, blood, or other intra-abdominal space-occupying lesions; and any conditions that limit expansion of the abdominal wall. Normal IAP is approximately 5-7 mmHg in critically ill adults. The concept of intra-abdominal perfusion pressure is analogous to cerebral perfusion pressure. Abdominal perfusion pressure (APP) is equal to mean arterial pressure (MAP) minus intra-abdominal pressure.

Indication

- Consider screening patients with risk factors for IAH or ACS.
  - Postoperative patients (abdominal surgery)
  - Abdominal trauma
  - Ventilated patients with additional organ failure
  - Other signs of ACS

* Email: sujanty@gmail.com

- Oliguria, hypoxia, hypotension, acidosis, mesenteric ischemia, ileus, elevated ICP
- High cumulative fluid balance

Contraindications

- IAP monitoring requires placement of a urinary bladder catheter and measurement in the supine position. Any contraindication for these would apply.

Preparation

- Equipment
  - Urinary catheter
  - Urine bag for drainage of urine
  - Two 3-way stopcocks
  - Luer lock connector
  - Pressure transducer and tubing
  - 50ml luer lock syringe
  - Sterile 0.9% sodium chloride
  - Clamp

Procedure

1. The patient should be placed in the supine position for measurement. If this is not clinically feasible, it is important to recognize that elevation of the head of the bed will result in a higher IAP. Ensure all subsequent readings are taken in the same position to prevent false changes in measurement.
2. Using aseptic technique, connect the urinary catheter to the drainage bag using the stopcocks. Attach the transducer to a 3-way stopcock. An infusion catheter is connected to another stopcock via pressure tubing.
3. Prime the transducer set and monitoring lines with 0.9% sodium chloride.
4. Adjust the height of the transducer so that the top of the stopcock is level with the mid-axillary line. Zero the transducer.
5. Clamp the drainage tube to the urine bag.
6. Fill the bladder with 1 mL/kg (maximum 25 mL) of 0.9% sodium chloride using a syringe. The volume of fluid in the bladder should be constant for each measurement.
7. Close the stopcock of the syringe and allow 30-60 seconds for equilibration. Obtain the mean pressure reading upon end expiration (this minimizes the effects of pulmonary pressures).
8. Urine flow into the drainage bag should be uninterrupted except during IAP measurement.

9. The abdominal blood flow should produce fluctuations in the waveform. Air in the system or kinking of the monitoring lines may dampen the waveform. Refer to invasive hemodynamic monitoring guideline for more information on waveforms.
10. At end of the measurement, return the patient's head of bed to 30° to reduce the risk of ventilator-associated pneumonia.

Complications

- Complications related to urinary catheter placement including urinary tract infection

Interpretation

- IAH grading
  Grade I IAP: 12-15 mmHg
  Grade II IAP: 16-20 mmHg
  Grade III IAP: 21-25 mmHg
  Grade IV IAP: > 25 mmHg
- If IAH exists on baseline assessment, perform serial IAP measurements throughout the patient's critical illness.
- ACS
  Sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction / failure

References

Al-Dorzi HM, Tamim HM, Rishu AH, Aljumah A, Arabi YM. Intra-abdominal pressure and abdominal perfusion pressure in cirrhotic patients with septic shock. *Ann. Intensive Care*. 2012;2 Suppl 1:S4.

Cheatham ML, Malbrain MLNG. Cardiovascular implications of abdominal compartment syndrome. *Acta. Clin. Belg. Suppl*. 2007;(1):98–112.

Cheatham ML, Safcsak K, Block EF, Nelson LD. Preload assessment in patients with an open abdomen. *J. Trauma*. 1999;46(1):16–22.

Cheatham ML, Malbrain MLNG, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. II. Recommendations. *Intensive Care Med*. 2007;33(6):951–962.

Cheatham ML, Safcsak K. Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival? *Crit. Care Med.* 2010;38(2):402–407.

Malbrain MLNG. Is it wise not to think about intraabdominal hypertension in the ICU? *Curr. Opin. Crit. Care*. 2004;10(2):132–145.

Malbrain MLNG, Cheatham ML, Kirkpatrick A, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. *Intensive Care Med*. 2006;32(11):1722–1732.

Malbrain MLNG, Chiumello D, Pelosi P, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit. Care Med.* 2005;33(2):315–322.

Malbrain MLNG, Chiumello D, Pelosi P, et al. Prevalence of intra-abdominal hypertension in critically ill patients: a multicentre epidemiological study. *Intensive Care Med*. 2004;30(5):822–829.

Oda S, Hirasawa H, Shiga H, et al. Management of intra-abdominal hypertension in patients with severe acute pancreatitis with continuous hemodiafiltration using a polymethyl methacrylate membrane hemofilter. *Ther. Apher. Dial*. 2005;9(4):355–361.

Santa-Teresa P, Muñoz J, Montero I, et al. Incidence and prognosis of intra-abdominal hypertension in critically ill medical patients: a prospective epidemiological study. *Ann. Intensive Care*. 2012;2 Suppl 1:S3.

Starkopf J, Tamme K, Blaser AR. Should we measure intra-abdominal pressures in every intensive care patient? *Ann. Intensive Care*. 2012;2 Suppl 1:S9.

Sun Z-X, Huang H-R, Zhou H. Indwelling catheter and conservative measures in the treatment of abdominal compartment syndrome in fulminant acute pancreatitis. *World J. Gastroenterol.* 2006;12(31):5068–5070.

Vidal MG, Ruiz Weisser J, Gonzalez F, et al. Incidence and clinical effects of intra-abdominal hypertension in critically ill patients. *Crit. Care Med.* 2008;36(6):1823–1831.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 13

Intra-Aortic Balloon Pumping (IABP)

Jason Bartock, MD[3], Carol Choe, MD[2]
and Sri Sujanthy Rajaram, MD, MPH[1*]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US
[3]Greater Baltimore Medical Center, Baltimore, MD, US

Introduction

The rate of death among patients with cardiogenic shock, usually as a complication of myocardial infarction (MI), remains high even in the setting of coronary revascularization.

The intra-aortic balloon pump (IABP) is the most widely used hemodynamic support device in these critically ill patients (US: class IB recommendation). The goal of the IABP is to increase myocardial oxygen supply and decrease myocardial oxygen demand, resulting in an overall improvement in ventricular performance.

An IABP supports hemodynamics through counterpulsation. Inflation occurs in diastole and deflation occurs in early systole. The balloon inflates immediately after aortic valve closure (diastole) and displaces blood to the proximal aorta. This leads to an increase in coronary blood flow and an improvement in systemic perfusion (Figure 1a). Systemic perfusion is improved by augmentation of the Windkessel effect (potential energy stored in the aortic root that is converted to kinetic energy by its elastic recoil). Inflation during diastole causes a diastolic augmentation wave form which results in an over all augmentation of the patients systolic blood pressure.

The balloon deflates just before aortic valve opening during systole causing a reduction in aortic volume through a vacuum effect, thereby reducing afterload (Figure 1b).

* Email: sujanty@gmail.com

Indications

- Acute MI with cardiogenic shock
- Acute mitral insufficiency or ventricular septal defect
- High risk percutaneous coronary intervention (PCI)
- Refractory unstable angina
- Weaning from bypass
- Left ventricular failure
- Ventricular arrhythmia post-MI

Contraindications

- Aortic insufficiency
- Aortic dissection
- Left ventricular failure with little hope for recovery
- Aortic stent
- Uncontrolled sepsis
- Large abdominal aortic aneurysm
- Severe peripheral vascular disease
- Significant aortic plaque

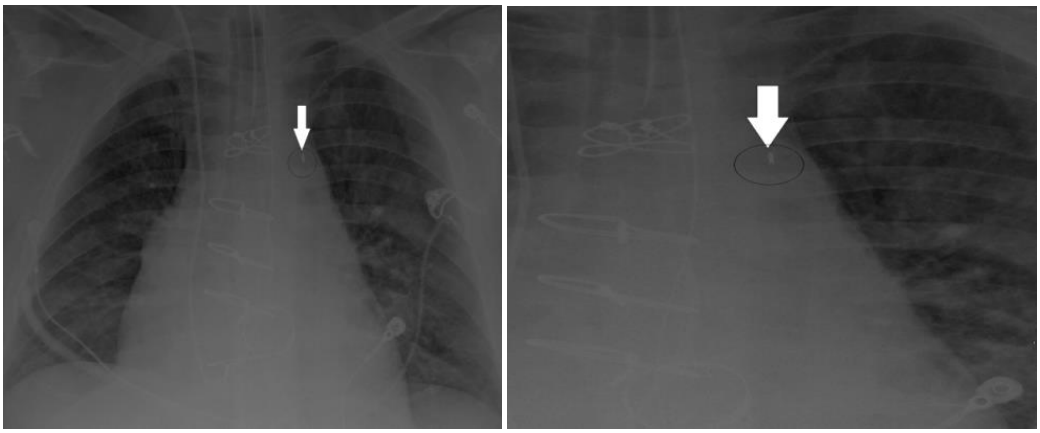
Preparation

The IABP device consists of two components:

- Flexible catheter with a single lumen that allows pressure monitoring and the delivery of helium gas (sizes range from 20-50cc)
- Mobile system console for cycle control and gas storage

Procedure

The intraaortic balloon is usually placed by a Cardiologist or Intensivist into one of the femoral arteries via Seldinger technique using the guidewire similar to placing a central venous line. After placement, a chest x-ray is obtained to confirm placement prior to connecting the catheter to the console. Catheter tip should be positioned between second and third intercostal space just below the level of the subclavian vein. The lowest portion of the inflated balloon should be above the level of the renal artery when postioned well. The system is purged with helium. A heparin infusion is initiated to maintain a partial thromboplastin time of 60-80 seconds or an ACT of 1.5-2.0 times normal.



CXR showing the tip of the Catheter.

Tip of the IABP catheter should be positioned between the second and third intercostal space about 2 cm distal to the subclavian artery. If it is above this level obstruction to the subclavian artery and if positioned below can have ineffective pumping with obstruction of the renal artery is a potential risk.

Interpretation

The trigger is set to correspond to the ECG and the arterial pressure waveforms

- ECG waveform
  - ✓ Inflates with the middle of the T-wave (diastole) and deflates with the peak of the R-wave (systole)
  - ✓ Poor ECG quality / arrhythmia can result in erratic balloon inflation
- Arterial pressure waveform (Figure 2)
  - ✓ Inflates just before the dicrotic notch at the onset of diastole and deflates just before the upstroke in the waveform during isovolmetric contraction phase of systole
  - ✓ Diastolic augmentation
    - Balloon inflation during diastole results in augmentation of the systemic blood pressure, which gives a second peak. Diastolic augmentation should be higher than the patient's own unassisted systolic pressure.

When the ballon is inflated it is 90% occlusive in the aorta. To recognize the inflation and deflation timing abnormalities IABP should be placed in 1:2 assist means that intial wave form shown in Figure 2 is an assisted arterial wave form followed by patient's own un-assisted wave form and again the next cycle is assisted.

Properly timed inflation occurs during diastole, just before the dicrotic notch, after the aortic valve closure during isovolumetric relaxation phase and gives a 'V' configuration in the waveform. This timed inflation gives an augmented diastolic pressure wave form which is higher than the patient's own systolic pressure and acts as an increased systemic blood

pressure. Goals of inflation are to increase coronary perfusion pressure, mycocardial oxygen supply, pulse pressure and systemic perfusion pressure.

Suboptimal timing of inflation and deflation of the balloon may result in hemodynamic instability and 4 types of errors can occur (Figure 3). Early Inflation and Late Deflation are potentially dangerous timing errors.

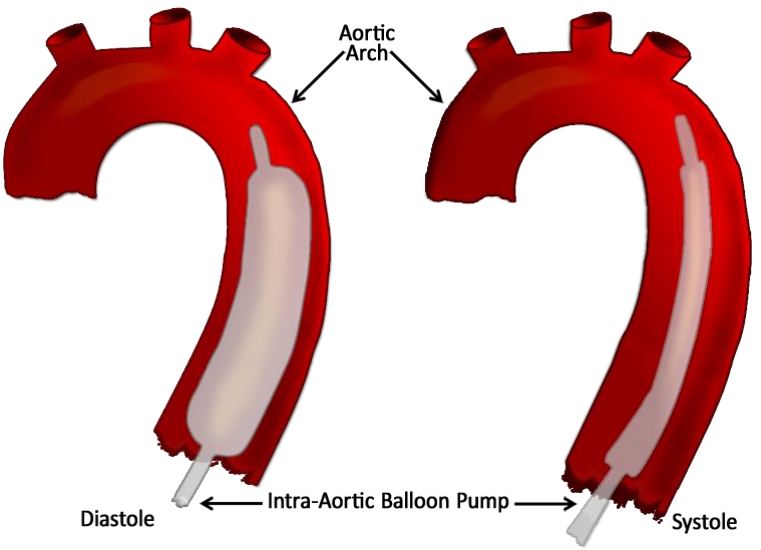


Figure 1a. Inflation

Figure 1b. Deflation.

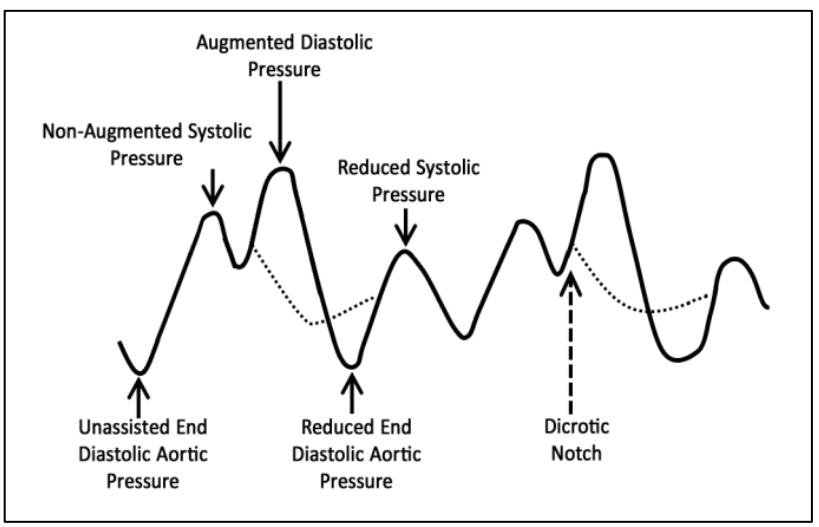
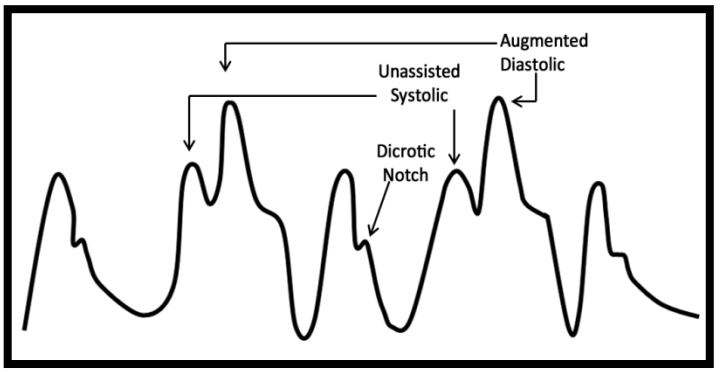


Figure 2. IABP Wave Form 1:2 Pump Assist.

Earlt Inflation: If inflated too early, the timimg can fall before the aortic valve closure during systole. This early inflation results in loss of forward outflow and loss of cardiac output which can result in significant hemodynamic instability.



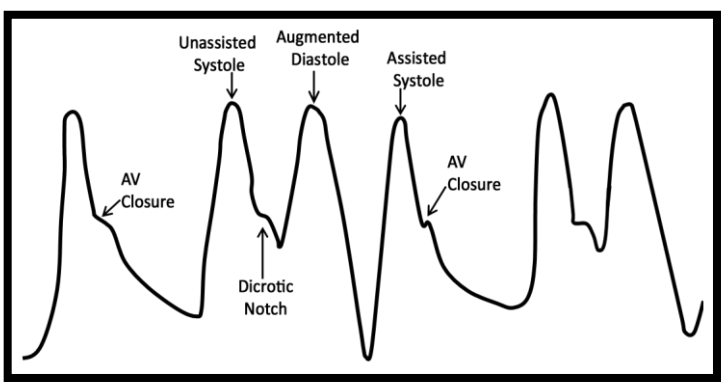
Wave form looses 'V' configuration, inadequate diastolic augmentation

Figure 3a. Early Inflation.

Early Inflation effects: Premature closure of the aortic valve, aortic regurgitation, increased left ventricular stress, increased oxygen demand and Lower diastolic augmentation.

Late Inflation: If the dicrotic notch is visible in the wave form means the inflation occurred late during diastole. As a result the diastolic augmentation is not high enough to provide adequate coronary perfusion.

Wave form with a visible dirotic notch



Late Inflation: Suboptimal coronary perfusion

Figure 3b. Late Inflation.

Properly timed deflation occurs during isovolumetric contraction phase of systole during PR interval which causes a fall in aortic pressure prior to the ventricular ejection. This corresponding low aortic pressure is the Ballon Assisted End Diastolic pressure (BAEDP), which is lower than the patient's own Aortic End Diastolic Pressure (AEDP). Lower BAEDP helps to lower the afterload and ventricle does not have to generate high pressure to eject the stroke volume. As a result of this lower BAEDP, the next Assisted Peak Systolic Pressure (APSP) is lower than the patient's own Peak Systolic Pressure (PSP). Goals of deflation are to decrese afterload and myocardial oxygen demand.

Early Deflation: If deflated too early APSP wave form is not lowered, reaches the same height as patient's own PSP results in suboptimal afterload reduction increased myocardial oxygen demand and angina may occur due to retrograde coronary flow.

Early Deflation effects: Suboptimal diastolic augmentation, afterload reduction and coronary perfusion.

Wave form assisted peak systolic pressure (APS) equal to patient's own PSP

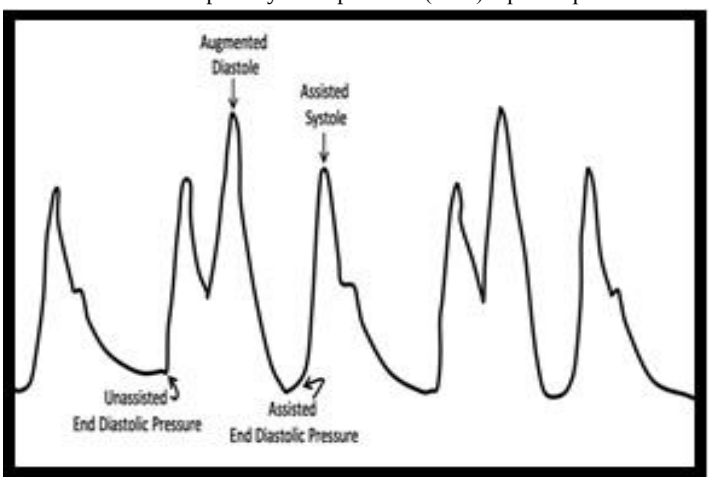


Figure 3c. Early deflation.

Late Deflation: IF ballon is deflated late the timing falls at the beginning of ventricular ejection. Ventricle has to eject the blood against an inflated ballon against resistence during the ascending limb of arterial waveform where 2/3 of the stroke volume delivery occurs. This results in increased ventricular work load and severe loss of stroke volume. Action should be taken immediately to prevent ventricular failure. This can be recognized by not having a lower BAEDP than patient's own AEDP.

Wave form BAEDP is not lower than patient's own AEDP

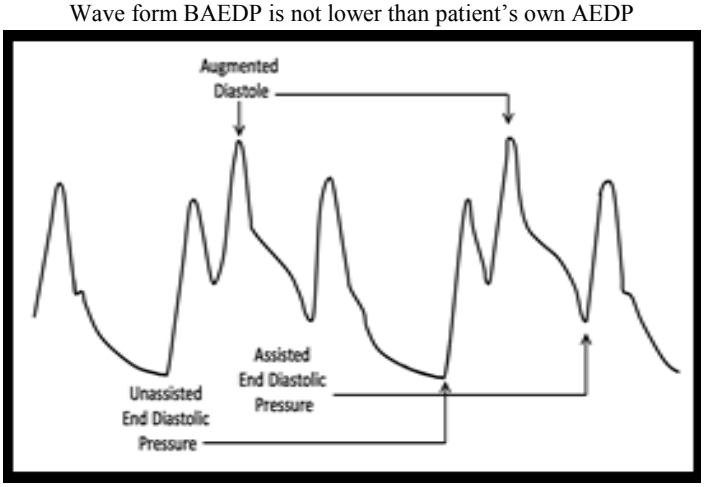


Figure 3d: Late Deflation.

Late deflation effects: Absent afterload reduction, increased oxygen consumption, impedes LV ejection and increases afterload. This is dangerous and causes significant hemodynamic instability

Weaning

Timing and speed of weaning is dictated by the patient's hemodynamic status. Weaning can be started when inotropic requirements are minimal. Weaning is usually accomplished by decreasing the frequency and or volume of the balloon.

- Weaning by decreasing frequency is accomplished by decreasing the frequency of assistance by one balloon inflation per cardiac cycle (1:2, 1:3, 1:4, 1:8)
- Weaning can also be accomplished by decreasing the amount of volume delivered to the balloon. Do not reduce the volume of the balloon more than 2/3 the capacity.

Throughout the weaning process monitor ECG, HR, BP, urine output, mentation, distal perfusion, and cardiac output.

IABP Support May be Discontinued If

- Absence of Angina
- Minimal inotropic agents
- No evidence of hypoperfusion, U/o > 30ml/hr
- HR<100bpm and with minimal ectopy (<6/min)
- CI >2l/min/m2 and didn't drop by more than 20%
- PCWP didn't increase more than 20%

** NEVER turn off in situ because of risk of thrombus formation**

Complications

- Limb ischemia
- Thromboembolism
- Compartment syndrome
- Aortic dissection
- Infection
- Tamponade
- Gas embolism
- Cholesterol emboli

References

Ferguson JJ 3rd, Cohen M, Freedman RJ Jr, et al. The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. *J. Am. Coll. Cardiol.* 2001;38(5):1456–1462.

Intra Aortic Balloon Pump Support: Lecture, Sri Sujanthy Rajaram, 2012, Division of Critical Care Medicine, Cooper University Hospital, Camden, NJ

Kern M.J, King SB. Cardiac Catheterization, Cardiac Angiography, and Coronary Blood Flow and Pressure Measurements. In Fuster V, Walsh RA, Harrington RA, EDS. *Hurst's The Heart.* 13th ed. New York, NY: McGraw-Hill; 2010.

Krishna M, Zacharowski K. Principles of intra-aortic balloon pump counterpulsation. *Continuing Education in Anaesthesia, Critical Care & Pain*. 2009;9(1):24–28.

Thiele H, Zeymer U, Neumann F-J, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. *N. Engl. J. Med.* 2012;367(14):1287–1296.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 14

Intracranial Pressure Monitoring

Michelle Ghobrial, MD[1], Jacqueline S. Urtecho, MD[1] and Jawad F. Kirmani, MD[2,•]

[1]Neuro-Critical Care, Thomas Jefferson University, Philadelphia, PA, US
[2]New Jersey Neuro Science Institute, JFK Medical Center, Edison, NJ, US

Introduction

When brain injuries occur, having a solid understanding of cerebral physiology becomes paramount in managing patients. The intracranial vault is a fixed volume comprised of brain, blood and cerebral spinal fluid. An increase in the volume of one component will cause a decrease in the volume of the other components, or a compensatory increase in pressure will occur in order to maintain equilibrium (Figure 1).

Intracranial pressure (ICP) is determined by subtracting cerebral perfusion pressure (CPP) from mean arterial pressure (MAP). CPP is the pressure driving blood through the vessels. In cases of elevated ICP or systemic hypotension, cerebral perfusion pressure decreases. Normal ICP varies with age and body posture but is generally considered to be 5–15 mmHg in supine adults. The un-injured brain has intact autoregulation and can make adjustments in vascular tone, reflected as changes in MAP, in order to maintain a normal CPP (50-70 mmHg) and cerebral blood flow. After brain injury, autoregulation becomes disrupted and small changes to MAP or ICP can have devastating consequences (Figure 2).

Prior to brain herniation, the patient may experience a myriad of symptoms including headache, nausea, vomiting, ocular palsy, altered consciousness, and papilledema. Vital signs also become unstable, resulting in a phenomenon known as Cushing's triad: systolic hypertension, bradycardia and respiratory irregularity. Intracranial monitoring is an

• E-mail: jkirmani@gmail.com.

invaluable resource used to guide therapies aimed at lowering ICP and preserving at-risk brain tissue.

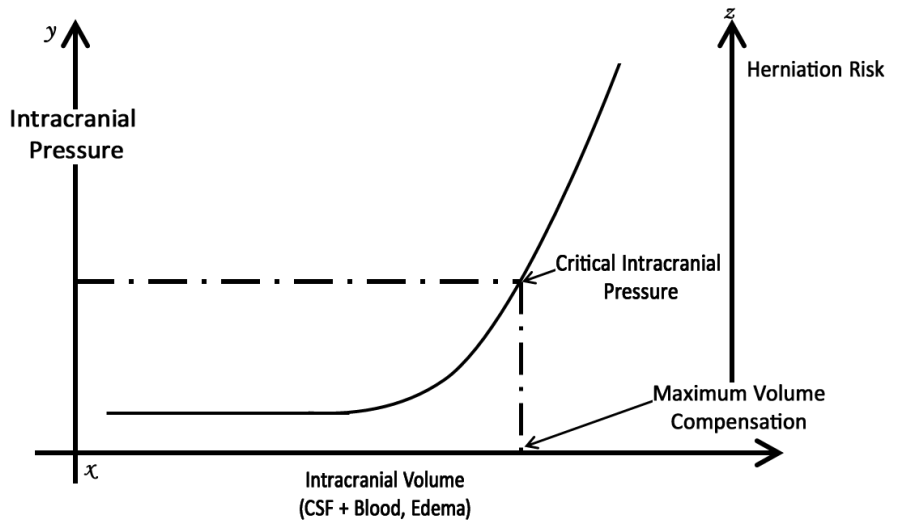


Figure 1. Intracranial pressure-volume curve demonstrating a compensatory phase followed by a critical threshold after which ICP dramatically rises.

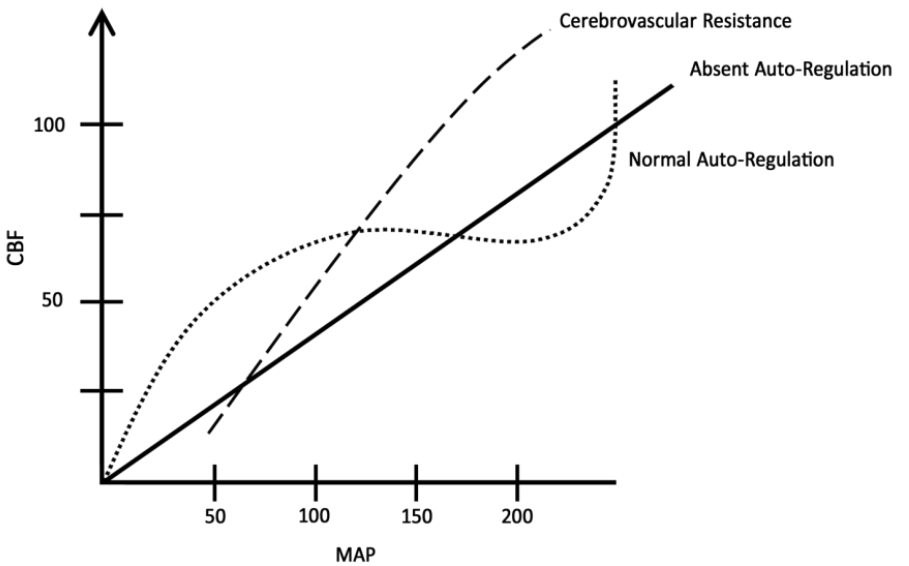


Figure 2. Cerebral autoregulation curve showing maintenance of cerebral blood flow within a wide MAP range in a normal individual. However, in the absence of this mechanism, a linear relationship may prevail.

Indications

- Acute hydrocephalus with clinical symptoms of intracranial hypertension (intracranial hemorrhage, mass, or any intracranial pathology causing malignant cerebral edema)
- Salvageable patients with severe traumatic brain injury (TBI) with Glasgow Coma Scale (GCS) score <8 and an abnormal computed tomography (CT) of the head.
- Severe TBI with GCS < 8 and normal CT head PLUS at least 2 of following: age >40 years old, abnormal motor posturing, systolic blood pressure <90 mmHg

Contraindications

- Coagulopathy or patients being treated with anticoagulants
- Scalp infection at or near the proposed site of placement
- Non-communicating hydrocephalus
- Inability to provide close supervision

Preparation

Determine the type of monitor to be used:

- External ventricular drains (EVDs) are the gold standard for ICP monitoring. A fluid-filled tubing is placed in a lateral ventricle and connected to an external transducer. This allows drainage of cerebral spinal fluid (CSF) in addition to measurement of intracranial pressures.
- Intraparenchymal monitors only measure pressure without draining CSF.
- Epidural, subdural, and subarachnoid pressure monitors are less accurate than intraventricular or intraparenchymal monitors. They do not allow for drainage of CSF.

Procedure

ICP monitoring devices are typically placed by a neurosurgeon in the operating room or at the bedside in the ICU.

Figure 3 denotes the locations of the various monitoring devices. If a drain is placed, the neurosurgeon or intensive care physician decides how to remove CSF.

There are two ways in which pressure can be monitored while CSF is being drained. One method is to keep the stopcock “open” and allow CSF to drain freely while performing intermittent ICP readings.

The major complication of this approach is the inability to control CSF drainage. Positioning of the patient is critical when this mode is utilized. This is usually tolerated and safe provided that the patient‘s caregivers are aware that the drain is open.

The other way to monitor ICP is through a closed system in which the stopcock is kept closed and is opened only when the ICP reaches a specific threshold. Some believe this is a safer way to drain CSF, as the patient is not at risk for accidental over-drainage of cerebrospinal fluid. However, it may be labor-intensive for a patient who requires frequent drainage.

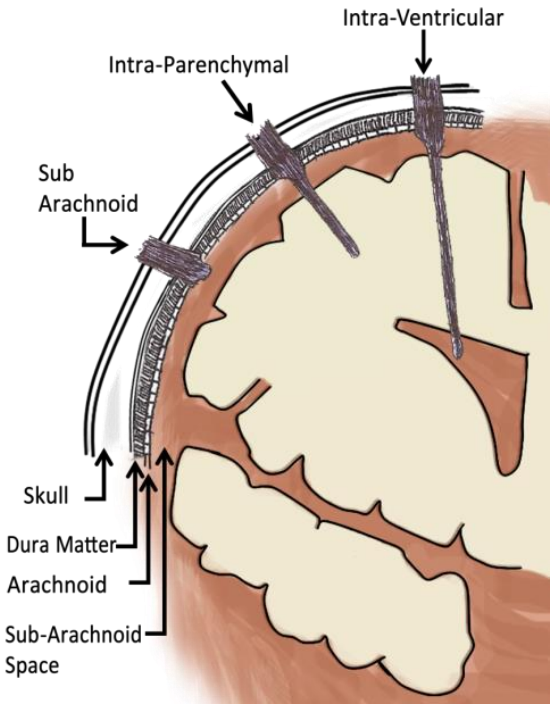


Figure 3. The placement of various types of intracranial monitors.

Complications

- Hemorrhage
- Bacterial colonization or clinical infection
- Malposition or malfunction of the catheter
- Obstruction of the ICP monitor and catheter
- Disconnection of transducer
- Over-drainage of CSF

Interpretation

Successful placement of an EVD should yield a characteristic waveform composed of three components (Figure 4a):

- P1 is the percussive wave (arterial systolic wave) and should have a sharp peak and relatively constant amplitude.
- P2 is the tidal wave and reflects brain compliance
- P3 is the dicrotic wave and represents aortic valve closure

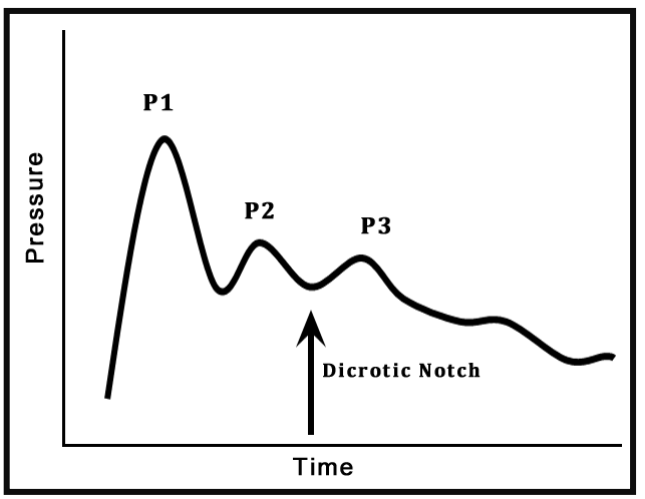


Figure 4a. Normal ICP waveform.

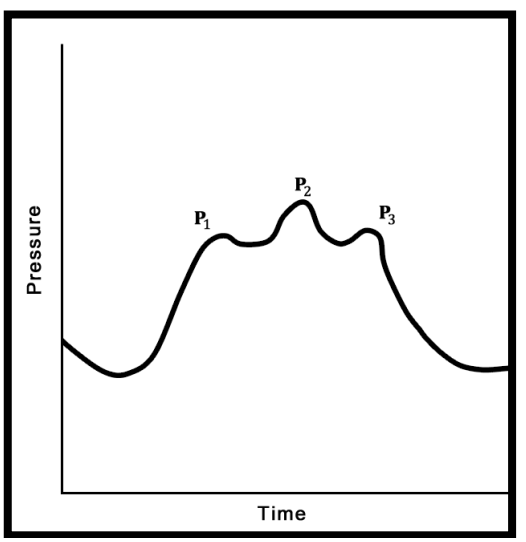


Figure 4b. ICP waveform with decreased ventricular compliance. Note P2 is higher than P1.

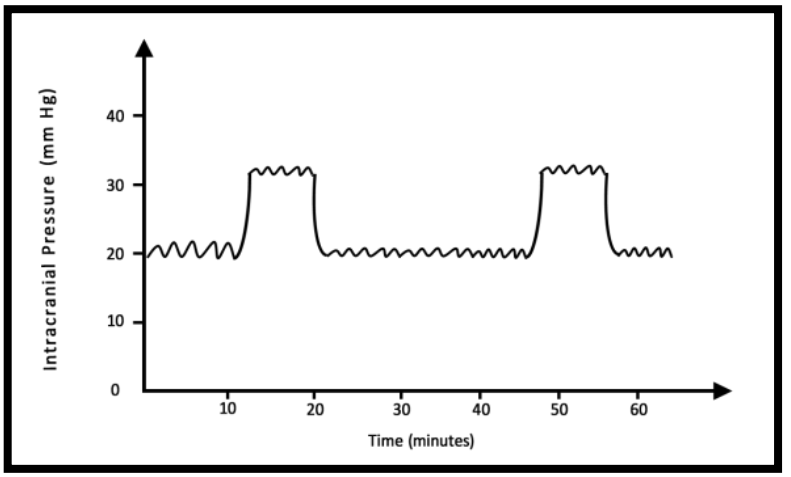


Figure 5. Lundberg A waves = Plateau Waves.

Table 1. ICP waveform for various intracranial pathologies

Conditions that can cause elevated ICP	Changes in the ICP Waveform
Mass lesions: tumor hematoma abscess	Increase in amplitude of P2 waveform (see Figure 4)
Increase in BP (Systemic hypertension)	Increase in amplitude of P1 waveform
Increase in CSF volume Hydrocephalus (obstructive or communicative) Overproduction	Increase/decrease in all waveforms but not individual components
Increased venous return Hypoventilation Increased venous compression	Increase in amplitude of P2 waveform

Alterations in the waveform can indicate intracranial hypertension and poor brain compliance (Figure 4b). Sustained elevated ICP lasting for 5-10 minutes are referred to as "Plateau Waves" and represent early brain herniation (Figure 5).

References

Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons; Joint Section on Neurotrauma and Critical Care, AANS/CNS: Guidelines for the management of severe head injury. *J. Neurotrauma,* 2007; 24(Suppl.):S-1–S106.

Greenberg, Mark. Handbook of Neurosurgery. 6th ed. New York: Thieme, 2006.

Holloway K. L., Barnes T., Choi S., et al. Ventriculostomy infections: the effect of monitoring duration and catheter exchange in 584 patients. *J. Neurosurg.,* 1996; 85(3):419–424.

Jallo J., Loftus C. Neurotrauma and Critical Care of the Spine. 1st ed. Thieme; 2009.

Lee, Kiwon. The NeuroICU Book. 1st ed. New York: McGraw Hill, 2012.

Paramore C. G., Turner D. A. Relative risks of ventriculostomy infection and morbidity. *Acta Neurochir. (Wien),* 1994; 127(1-2):79–84.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 15

Intubation and Airway Monitoring

Munira Mehta, MD[1], Mahesh Bhagat, MD[2],
Yong-Bum Song, PharmD, BCPS[2]
and Sri Sujanthy Rajaram, MD, MPH[2]
[1]Mercy Fitzgerald Hospital, Darby, PA, US and
Nazareth Hospital, Philadelphia, PA, US
[2]JFK Medical Center, Edision, NJ, US

Introduction

Critical Care physicians often deals with airway emergencies and intubations. Assessment of the airway should be quick. Thick short neck, crowded oral airway, micrognathia and anatomical anomalies of the chin may cause difficult airways.

LEMON rule is described in evaluation of difficult airway.
L- Look externally for an obvious signs of difficult airway
E- Evaluate 3:3:2 rule

- Do 3 fingers fit between incisors when mouth is wide open? – If yes, then temporomandibular joint mobility is good.

- Is the distance between the mentum and hyoid bone 3 fingers? – If yes, then it is good length of mandible. More or less can make bag mask ventilation or intubation difficult.

- Is the distance between the hyoid bone and thyroid 2 fingers? – If yes, then length of the neck is good.

M- Mallampati score
O- Obstruction or obesity,
N- Neck mobility

Modified Mallampati classification is described in assessment of airway in cooperative awake patient. During an emergent intubation in Intensive Care Unit (ICU) patients, one can get an assessment by opening the patient's mouth after sedation.

Class 0: Open the mouth, if able to protrude the tongue out, see visible epiglottis.

Class 1: Visible Soft palate, Fauces, Uvula, Pillars

Class 2: Pillars not visible, Visible Soft palate, Fauces and Uvula

Class 3: Pillars and fauces not visible, Visible soft palate and base of uvula

Class 4: Soft Palate not visible

During direct laryngoscopy view of the epiglottis is essential for proper endotracheal tube placement.

Cormack-Lehane view is described often and widely used.

Grade 1- visible glottis

Grade 2 – Half of glottis is visible

Grade 3 – Only epiglottis is visible

Grade 4- No laryngeal structures are visible

Obviously Malampati class 4 and Cormack- Lehane view Grade 4 are difficult airways.

Indication for Intubation (for Intensivists)

1. Airway protection (loss of gag or cough reflexes): to prevent aspiration
    a. Stroke
    b. Status epilepticus
    c. Head injury
    d. GCS < 8 or poor mental status
    e. Massive hematemesis and hemoptysis
2. Airway obstruction
    a. Acute laryngeal edema
    b. Angioedema
    c. Intrinsic or extrinsic compression of airway from tumors
3. Anticipated loss of airway control
    a. Respiratory distress
    b. Stridor
    c. Expanding neck hematoma
    d. History of neck injury
    e. History of burns and smoke inhalation (presence of soot in mouth, lips or nares)
    f. Progressive increase in the work of breathing
4. Failure to ventilate: evident by Co2 retention and acidosis
    a. CNS: stroke, status epilepticus, drug overdose, head injury, general anesthesia.
    b. Respiratory muscle weakness – myasthenia gravis, GBS, flail chest.
    c. Airways – asthma, COPD exacerbation.

5. Failure to oxygenate: evident by worsening hypoxia and increasing alveolar arteriolar gradient
    a. Pneumonia
    b. ARDS
    c. CHF
    d. Pulmonary edema
    e. Pulmonary emboli
    f. V/Q mismatch
6. Intubation for surgical procedures in the operating room- Often elective intubation

Contraindication

No absolute contraindication. Facial fracture is a contraindication for nasal intubation. In patients with unstable neck and cervical spine instability need to be cautious.

Challenges in Airway Management in ICU

As compared to airway management for elective surgeries in OR, airway management in ICU is different, challenging and more unpredictable.

1) Most intubations are not planned, therefore we may not get a chance to assess airway in advance and prepare accordingly.
2) Keep in mind loose teeth, dentures, edentulous, obese or short neck patients that can make intubation difficult.
3) Patients may already have underlying disease causing severe hypoxia. After giving induction agents there may be further desaturation. Ensure patient can be oxygenated adequately by opening the airway.
4) Patients could have active ongoing hematemesis, vomitus or copious respiratory secretions, thus making airway more difficult and high risk of aspiration, particularly after food intake.
5) Critically ill patients are already hemodynamically unstable and induction agents and positive pressure ventilation can further make them hypotensive and more unstable.
6) Patients with already labored breathing or respiratory muscle fatigue, can further decrease or lose their respiratory drive and reserve after induction agents.
7) Unplanned extubations or blocked or kinked endotracheal tubes add further complexities by either having to re-intubate or change endotracheal tubes when the patient is already hypoxic and or hemodynamically unstable
8) Always plan and prepare for difficult intubation during airway management in ICU.

Preparation and Procedure

- Anticipate difficult airway in advance and ASK for HELP sooner than later.
- It is necessary to have a team including nurse, respiratory therapist, and an assistant who helps with all equipment. Know your back up help is always an Anesthesiologist or Colleague.
- Quickly check and prepare all equipment while bagging and oxygenating the patient. Be in charge at the head of the bed.
- If difficult airway is anticipated – it is extremely important to alert a specialized team –usually anesthesiologist.

Remember the SOAP format.

1. **S**uction – Using Yankauer suction catheter clear all the secretions.
2. **O**xygen – Make sure O2 is turned on and connected. Use Ambu bag to pre oxygenate. Use of Positive End Expiratory Pressure (PEEP) valve in the Ambu bag improves oxygenation. PEEP of 5 to 20 can be used.
3. **A**irway equipment and **P**osition –
    a. Adjust the height of the bed and position the patient. Keep doughnut pillows or other pillows or blankets or roll towels to position.
    b. Attach the direct laryngoscope to either a curved Mac 3 or Mac 4 blade. Keep straight blades like Miller-2 or Miller-3 blades available if required.
    c. If your institute has Glidescope, keep it ready to use at the bedside. Glidescope has its own single-use laryngoscope blades (size 3 or 4 is commonly used) and specific stylet. Plug it in electric socket so as to get the light source and ensure the video screen is working.
    d. Use size 7.5 or 8.0 cuffed endotracheal tube (ETT) in adults. Size 8.0 mm tube helps for suctioning and bronchoscopy if needed. Check for cuff leak with 10-15 ml of air and deflate the cuff. Place the stylet in place. You can curve the stylet to make the entry easy.
    e. Use oral airway if biting or difficulty encountered during bagging.
    f. An End Tidal CO2 monitoring device should be used to confirm placement of the endotracheal tube – either calorimetric or waveform capnography device. Change of color from purple to yellow indicates tracheal intubation. Always check placement of ETT by listening for breath sounds in both lung fields and abdomen to ensure no esophageal intubation took place. You must also confirm placement by an immediate chest x-ray and rule out pneumothorax. Correct position of the ETT should be approximately 2-3 cm above the level of the carina and below the level of the clavicle. Direct laryngoscopy can be used to check placement if difficulty encountered with oxygenation.
    g. Endotracheal tube should be secured using comfit, silk tape and Anchor fast (Oral endotracheal tube fastener), prior to connecting to the ventilator.
4. **P**harmacy or Medications:

Commonly used agents are for rapid sequence intubation (RSI) are induction agents like etomidate and propofol, opiates like fentanyl to control pain and discomfort, benzodiazepines like midazolam as sedatives and muscle relaxants like depolarizing neuromuscular blocking agent (NMB) succinylcholine or non-depolarizing neuromuscular blockers like rocuronium and vecuronium. Rapid Sequence Intubation (RSI) is very often practiced in the emergency rooms and ICUs to reduce the risk of aspiration. During RSI sedatives and neuromuscular blockers are simultaneously used. When paralytics are not used, patient still has the intrinsic drive to breathe. During inhalation airway closes and we cannot pass the ETT. But using adequate sedation and analgesia, during exhalation when the glottis opens up, vocal cords become visible and ETT can be passed through the cords swiftly.

Table 1. Medications Used during Intubation

Medication	**Dose**	**Time to effect**	**Duration of action**	**Side effects**	**Points**
Etomidate (Sedative)	0.3 mg/kg	15-45 seconds	3 -12 minutes	Bronchospasm Adrenal Insufficiency Lowers seizure threshold Causes myoclonic jerks	Useful in hypotensive patients Good for elevated ICP, Decreses cerebral blood flow & metabolic rate
Propofol (Amnestic Sedative)	1-2 mg/kg	15-45 seconds	5-10 minutes	Hypotension Bradycardia Respiratory depression Decrease in cardiac contractility Unexplained EKG changes particularly continuous infusion	Consider in hemodynamically stable patients Useful in patients with elevated ICP and head injuries
Ketamine (Analgesic Amnestic Sedative)	1-2 mg/kg	30 seconds	5-15 minutes	Bronchodilation Agitation Hypersalivation Hypertension Tachycardia Increase in ICP (ICP- conflicting data)	Good field anesthesia, does not cause respiratory suppression Good agent for reactive airway disase
Fentanyl (Analgesic)	1-2 mcg/kg	30 seconds	30-60 minutes	Hypotension Bradycardia Rigid chest syndrome (spasm of the respiratory muscles leading to respiratory depression or apnea)	Control pain, Discomfort Blunts catecholamine surge from stress

Table 1. (Continued)

Medication	Dose	Time to effect	Duration of action	Side effects	Points
Midazolam (Sedative Anxiolytic Amnestic)	1-2.5 mg	3-5 minutes	Less than 2 hours	Hypotension Cardiac arrhythmia Respiratory depression Amnestic effect (profound in elderly)	Sedation, Minimize pharyngeal & Laryngeal reflexes Faster onset of action if administered concurrently with opiods
Methohexital (Anesthetic)	1.5 mg/kg	< 30 seconds	10-20 minutes	Hypotension Spasmodic movements Seizure Cardiorespiratory arrest	Consider in hemodynamically stable patients Useful in patients with elevated ICP and head injuries
Rocuronium (Non-depolarizing neuromuscular blocker)	0.45 -0.6 mg/kg	1-2 minutes	30-67 minutes	Anaphylactoid reaction Cardiac arrhythmia Hpertension Hypotension	Longer duration of action as compared to succinylcholine Prolonged effect in hepatic disease
Vecuronium (Non-depolarizing neuromuscular blocker)	0.08-0.1 mg/kg	2-4 minutes	20-60 minutes	Respiratory insufficiency or apnea Hypersensitivity reactions associated with histamine release	Prolonged effect in hepatic disease Active metabolite may accumulate in renal insufficiency
Succinylcholine (Depolarizing neuromuscular blocker)	1.5 mg/kg	30-60 seconds	4-6 minutes	Hyperkalemia Cardiac arrhythmia Malignant hyperthermia May cause elevated ICP	Avoid if hyperkalemia present Prolonged effect in hepatic disease

Types of Intubation

1) Orotracheal intubation
2) Nasotracheal intubation

As an intensivist, orotracheal intubation is mostly commonly performed for critically ill patients.

Occasionally when difficult airway is encountered in an awake patient, nasal intubation can be performed by an experienced Intensivist. Use a small size endotracheal tube, lubricate well and insert the tube through the nose and listen for air flow at the end of the tube. If oral

airway is obstructed nasal intubation can be successful for an emergent airway. Obstruction of the nasal passage can result in sinus infection and leaving in too long is not recommended.

Orotracheal Intubation

After adequate patient positioning and preparation, pre oxygenate with 100 % oxygen for 2 to 3 minutes. Administer iv induction agents like propofol or etomidate, along with iv fentanyl and or iv midazolam. Key for intubation is adequate ventilation and maintaining the saturation above 90% all the time to avoid any hypoxia. Everyone should be able to maintain adequate saturation 100% of the time. If the patient is not ventilated well, cyanosis or bradycardia can occur and eventually patient can have cardiac arrest. Occasionally can use a Laryngeal Mask Airway(LMA) for ventilation in difficult situation. Even if difficulty encountered as long as you can bag the patient and ventilate well, help can arrive on time. Don't panic.

Position of the Patient

Sniff position is good to get good visualization of the vocal cords. Sniffing positions involves atlanto-occipital extension with elevation of head by 3 to 7 centimeters, causing flexion of the lower cervical spine and upper thoracic spine. Basically you are trying to align the three axes (Figure 1).

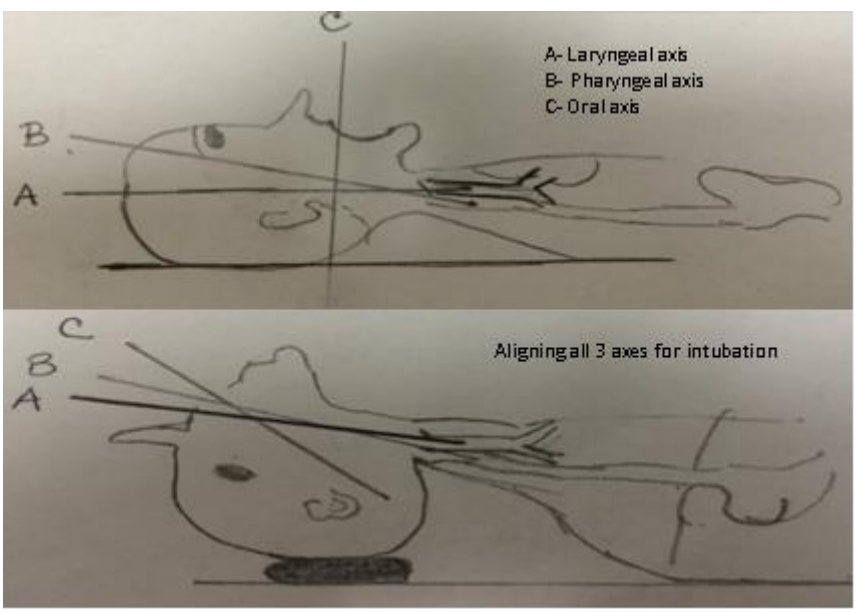


Figure 1.

Procedure

1. Maneuvers which can be used to keep the airway patent while under anesthesia.
    a. **Head-tilt chin-lift maneuver** lifts the tongue from the back of the throat and maintains an open, patent airway. But this maneuver can only be used if there is no concern for cervical spine injury.
    b. **Jaw thrust maneuver**: if cervical spine injury is a concern – the jaw thrust maneuver is a good technique to maintain a patent airway. In supine position, the mandible is displaced forward by physically pushing the angle of the mandible upwards using both thumbs. This helps lift the tongue forward and does not let it obstruct the airway.
2. Then administer the paralytic agent with sedatives if using RSI. Muscle relaxation keeps the vocal cords open and abducted and easy to pass the ETT. If not paralyzing wait until the vocal cords to abduct during exhalation and pass the ETT through the cords.
3. Scissors Maneuver: Open the mouth using the scissor motion of the fingers of the right hand i.e by placing the thumb on the lower jaw teeth and the index finger on the upper jaw teeth and applying a firm pressure on both jaws so as to open the mouth. The rotation and sliding movements of the temporo- mandibular joint are used to achieve maximal mouth opening.
4. Hold the laryngoscope handle with your left hand and insert the blade from the right angle of the mouth of the patient. Slide the laryngoscope blade in the mouth of the patient from the right angle of mouth to the center, sweeping the tongue towards the left. Keep the tongue always under the laryngoscope blade.
5. Once in the center, insert the blade slightly deeper into the pharynx towards the vallecula.
6. Identify the epiglottis and lift it up and adjust the tip of the laryngoscope blade in the vallecula. This is done by lifting the laryngoscope upwards towards the handle.
7. If cords are too anterior, bimanual laryngoscopy needs to be done. After the mouth is open and the laryngoscope blade is inserted properly as mentioned above, the right hand is then placed on the thyroid cartilage and it is externally manipulated either pressing downward or sideways to get the glottis into view. Once vocal cords are seen well ask an assistant to keep it steady in that spot.
8. Then use the right hand to hold the endotracheal tube such that the concave curve of the endotracheal tube faces away from you. Insert the endotracheal tube into the mouth, tip first, lateral to the laryngoscope blade and follow the curve of the palate. At all times, maintain the line of vision of the glottis and vocal cords. Do not let the endotracheal tube block your view of vocal cords.
9. Advance the endotracheal tube in enough so that the tip and the bevel pass beyond the vocal cords.
10. Ask an assistant to retract the stylet and remove it. This prevents any trauma to the soft tissues and blockage of the tube.
11. While the assistant is removing the stylet, the physician should hold the endotracheal tube steady in place and always maintain the view of the vocal cords and endotracheal tube through it.

12. Once the stylet is out, advance the endotracheal tube so that the cuff lies below the vocal cords and endotracheal tube rests at approximately 21 cm at the front incisors.
13. Inflate the cuff with about 5 to 8 ml of air.
14. Remove the laryngoscope blade out
15. Attach the end tidal-CO2 monitor and auscultate to confirm placement of the endotracheal tube.
16. Confirmation of the placement of endotracheal tube is done by several means
    a. The mist of air is seen in the tube
    b. Auscultation of lungs
    c. End-tidal CO2 monitor
    d. Chest x-ray
17. Before chest x-ray secure the endotracheal tube and connect to ventilator.
18. If difficult airway present consider using bronchoscopy or fiber optic bronchoscopy

Complications

Immediate complications:

1. Hypoxia.
2. Loss of airway: unable to oxygenate and unable to ventilate.
3. Hemodynamic instability.
4. Hypotension
5. Hypertension.
6. Tachycardia.
7. Arrhythmias.
8. Bronchospasm.
9. Laryngospasm.
10. Esophageal intubation.
11. Aspiration of loose teeth, oral contents, oral or tracheal secretions, gastric contents.
12. Trauma to structures: teeth, tongue, lips, eyes, corneal abrasion, in the soft tissues of oropharynx, larynx, vocal cords and trachea.
13. Trauma to neck and cervical spine, especially in patients with cervical spine injury or severe rheumatoid arthritis.
14. Dislocation of the temporomandibular joint during forceful opening of the mouth.

Long-term complications:

1. Tracheomalacia
2. Laryngomalacia
3. Tracheal stenosis
4. Injury to vocal cord

References

Godwin SA, Burton JH, Gerardo CJ, et al. Clinical Policy: procedural sedation and analgesia in the emergency department. *Ann Emerg Med.* 2014;63:247-58

Mechanical ventilation Manual, edited by Suhail Raoof, MD, Faroque A. Khan, MB

Micromedex. Truven Health Analytics, Inc. 2014. Accessed 4.8.2015

Miller's Anesthesia, 7th edition. Chapter 50. Airway Management in the Adult. John Henderson, pages 1573 - 1610

Stollings JL, Diedrich DA, Oyen LJ, Brown DR. Rapid-sequence intubation: a review of the process and considerations when choosing medications. *Ann Pharmacother*. 2014 Jan;48(1):62-76

www.medscape.com Accessed 3.31.2015

www.uptodate.com Accessed 3.31.2015

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter16

Jugular Venous Oxygen Saturation

Lauren Ng, MD[1], M. Kamran Athar, MD[1] and Mohammad Moussavi, MD[2*]

[1]Neuro- Critical Care, Thomas Jefferson University, Philadelphia, PA, US
[2]New Jersey Neuro Science Institute, JFK Medical Center, Edison, NJ, US

Introduction

Jugular venous oxygen saturation ($SjvO_2$) is an indicator of the balance between global cerebral blood flow (CBF) and cerebral metabolic demand ($CMRO_2$). It is measured to detect cerebral hypo perfusion or hyper perfusion to prevent or treat secondary brain injury.

Jugular venous oxygen saturation ($SjVO_2$) monitoring may evaluate global brain oxygen delivery and consumption, hence providing direct or indirect information and possible thresholds for detecting brain oxygenation, which may assist in evaluating brain hypo perfusion or hyper perfusion. The key factors affecting the SJVO2 include but not limited to the oxygen saturation of supplying arteries (SaO2) to the brain, hemoglobin concentration, and global and focal cerebral blood flow. Cerebral blood flow is mainly depending on cardiac index, peripheral resistance and blood viscosity.

In normal circumstances the brain tissues extract certain ratio of oxygen from the arterial oxyhemoglobin. Depending on demand and supply this ratio may vary between 25 to 50% under normal circumstances. Thus in a normal person, with greater than 95 % arterial SaO2, depending on the level of brain activity, the Jugular SJVO2 is usually expected to fluctuate between 50-80% . Values less than 50 % may indicate low oxygen supply and or high consumption. If SJVO2 is more than 80%, suggests that there may be low brain oxygen consumption in conditions such as brain infarction, low metabolism as in hypothermia,

* Email: mmoussavi@jfkhealth.org

hyperemia and in traumatic brain injury (TBI) patients. Many studies have shown secondary brain injury and worse outcome when the SJVO2 falls below or exceeds these two limits. In general if the value of SJVO2 drops less than normal the risk of ischemic event is higher and if it exceeds 80% the risk of brain edema and intracranial hypertension is high.

Indications

- Severe traumatic brain injury (TBI)

Jugular venous oximetry should be performed in patients with head trauma with Glasgow Coma Score (GCS) ≤ 8 where frequent episodes of cerebral desaturations can occur within the first 48 hours. In a study by Robertson et al, there was a strong association between cerebral desaturations and poor neurological outcome in adults with GCS ≤ 8 that was noted 5-10 days after TBI.

- Subarachnoid hemorrhage (SAH)

$SjvO_2$ may also have utility in SAH for prediction of vasospasm. $SjvO_2$ value in poor grade SAH may decrease prior to development of clinical intracranial vasospasm which potentially can be helpful in detecting and monitoring generalized vasospasm

- Cardiopulmonary bypass

While $SjvO_2$ has generally been reserved for use in brain injury, it has recently been used in surgeries involving cardiopulmonary bypass during which there is potential for desaturation associated with low mean arterial pressures, low hematocrit and rapid rewarming.

Contrindications

- Cervical spine injury
- Tracheostomy
- Coagulopathy
- Inability to tolerate Trendelenburg position

Preparation

There is much debate over which side should be cannulated – the dominant side or the injured side. In general, the consensus is to place the catheter on the dominant side.

The dominant side is determined either by occluding the jugular veins one at a time and seeing which side has the greater rise in intracranial pressure (ICP), by measuring the jugular foramina and assuming the larger one has more flow, or by using ultrasound to determine which side is larger. Nevertheless about +/- 5 differences in right or left side SjvO2 value is not abnormal.

Procedure

1) Once the dominant side has been established, the patient should be placed in Trendelenburg position or as horizontal as possible trying to prevent cerebral perfusion pressure (CPP) drops <60.
2) The internal jugular vein should be cannulated either distally between the two heads of the sternocleidomastoid muscle or proximally at the level of the cricoid. Cannulation should be towards cephalic direction.
3) A J-shaped guide wire is then advanced no more than 2-3 cm beyond the needle insertion site and the catheter is inserted until resistance is met. It is then withdrawn 0.5-1 cm to avoid cephalic vascular impact and injury. Ideal placement of the tip of catheter is at the level of C1/C2 to avoid extra cranial venous drainage when obtaining samples.

Placement should be confirmed with either a lateral neck roentgenogram (x-ray) or anterior-posterior (AP) neck x-ray (Figure 2). In the lateral x-ray, the catheter should end above the disc of C1/C2 and should be as close to the skull base as possible, at the level of the mastoid air cells. On the AP view, the catheter should lie cranial to a line extending from the atlanto-occiptal joint space to a line connecting the tips of the mastoid process, and caudal to the lower margin of the orbit.

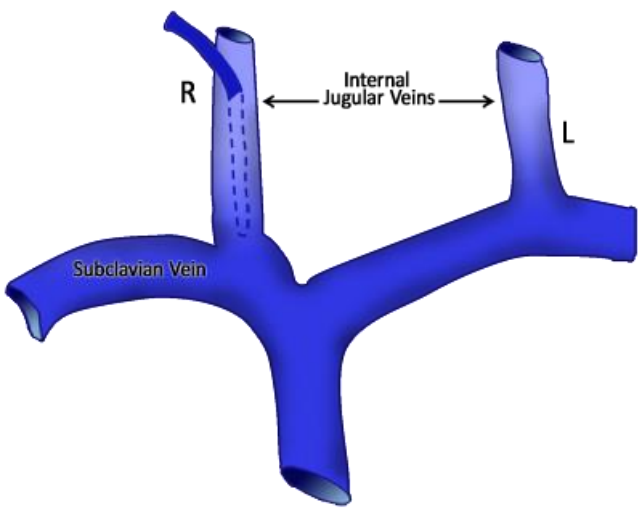


Figure 1. Placement of the Jugular Venous Oxygenation catheter.

Complications

- Carotid artery puncture
- Thrombosis
- Pneumothorax
- Nerve injury and infection

- Increased ICP secondary to decreased venous return, although this is thought to be very rare

Interpretation

- Normal $SjvO_2$ is around 55-70%. Values < 50% are associated with cerebral hypoxia, < 20% indicates irreversible cerebral injury and > 75% indicates hyperemia especially in traumatic brain injury (TBI) patients.
- $SjvO_2$ is inversely related to the cerebral arteriovenous oxygen difference ($AVDO_2$), which is a measure of oxygen extraction by cerebral tissue.
  - ✓ $AVDO_2$ <4 indicates that the oxygen supply is greater than demand
  - ✓ $AVDO_2$ >4 indicates ischemia with high oxygen extraction and inadequate CBF
- The main limitation of jugular venous oxygen saturation is that it is a global monitor and may not detect regional ischemia or hyperemia unless there is a large volume of tissue affected. Additionally, there may be inaccuracies in measurement from discrepancies between the left and right internal jugular veins, from the catheter being impacted against the vessel wall, thrombosis on the catheter tip and contamination from extracranial blood.

Table 1. Limitations of Jugular Venous Oximetry

Limitation	Rationale	Management
Incomplete mixing	Venous sample may not be representative of the entire brain if asymmetric venous drainage.	Cannulate the dominant internal jugular vein (usually right), or place on side of the most severe focal injury.
Extracerebral contamination	≈3% of jugular blood is contaminated by blood from scalp, meninges, and skull.	Radiograph confirmation. Location of catheter tip above lower border of C1 and withdraw sample slowly (<2 mL/min).
Bohr effect	Falsely high $SjvO_2$ values may occur from a leftward shift of the oxy-hemoglobin dissociation curve during alkaline conditions.	Detect by measuring low jugular bulb PO_2 (<27 mmHg)
Global measure	With focal cerebral injuries, $SjvO_2$ may not provide information about regional injury.	Measurement of arteriovenous lactate may be helpful as an indicator of anaerobic metabolism.
Insensitive to infratentorial flow	Brain stem and cerebellum contribute little to the venous outflow from the brain.	Is of limited value for monitoring patients with brainstem injuries.
Monitoring errors	Catheter may be against wall of the vein, coiled back on it self, or have fibrin formation on its tip.	Reposition the catheter, recalibrate the fiberoptic catheter, or instill 3mL/hr of heparinized saline.
$SjvO_2$ = jugular venous oxygen saturation.		

References

Barazangi N, Hemphill JC 3rd. Advanced cerebral monitoring in neurocritical care. *Neurol. India*. 2008;56(4):405–414.

Dhawan V, DeGeorgia M. Neurointensive care biophysiological monitoring. *J. Neurointerv. Surg*. 2012;4(6):407–413.

Dunn IF, Ellegala DB, Kim DH, Litvack ZN, Brigham and Women's Hospital Neurosurgery Group. Neuromonitoring in neurological critical care. *Neurocrit. Care*. 2006;4(1):83–92.

Fandino J, Kaku Y, Schuknecht B, Valavanis A, Yonekawa Y. Improvement of cerebral oxygenation patterns and metabolic validation of superselective intraarterial infusion of papaverine for the treatment of cerebral vasospasm. *J. Neurosurg.* 1998;89(1):93–100.

Macmillan CS, Andrews PJ. Cerebrovenous oxygen saturation monitoring: practical considerations and clinical relevance. *Intensive Care Med*. 2000;26(8):1028–1036.

Schell RM, Cole DJ. Cerebral monitoring: jugular venous oximetry. *Anesth. Analg.* 2000;90(3):559–566.

Wartenberg KE, Schmidt JM, Mayer SA. Multimodality monitoring in neurocritical care. *Crit. Care Clin*. 2007;23(3):507–538.

White H, Baker A. Continuous jugular venous oximetry in the neurointensive care unit--a brief review. *Can. J. Anaesth.* 2002;49(6):623–629.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 17

Lumbar Puncture

Jessica Mitchell, MD[2], Fiorella Nawar, MD[3] and Sri Sujanthy Rajaram, MD, MPH[1*]

[1] Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2] Univesity of New Mexico, Albuquerque, NM, US
[3] Sparks Regional Medical Center, Fort Smith, AK, US

Introduction

Lumbar puncture is a procedure performed either for therapeutic or diagnostic purposes. The main objective of the procedure is to remove a sample of cerebrospinal fluid from the subarachnoid space.

Indications

- Diagnostic
  - Infectious
    - Non-traumatic subarachnoid hemorrhage
    - Inflammatory process
    - Neoplastic process
    - Metabolic process
- Therapeutic
    - Administration of intrathecal medications
    - Relieve intracranial pressure (ICP)

* Email: sujanty@gmail.com

Contraindications

- Elevated ICP associated with an intracranial space occupying lesion
- Coagulopathy
- Overlying soft tissue infection

Preparation

Equipment (Lumbar Puncture kit)

- Skin cleanser (betadine or chlorhexidine solution)
- Sterile drape and sterile gown
- 20 gauge or smaller spinal needle with stylet
- Stopcock
- Manometer
- Specimen tubes
- 1% or 2% lidocaine WITHOUT epinephrine

Patient position

- Lateral decubitus position (Figure 1)
  - Patient in fetal position with knees and chin to chest
  - Back arched to keep body in a "C" position
  - Shoulders and hips aligned perpendicular to body
- Upright position
  - Head and shoulders bent forward
  - Lower back arched

Procedure

1. Visualize a line between the superior aspects of the iliac crests. This is the L3-L4 vertebral interspace.
2. Mark the L4 level and clean a large area around it in a widening concentric, circular manner.
3. Place sterile draping over this area and infiltrate the desired site with local anesthesia. Anesthetize the deeper tissues along the proposed tract of spinal needle insertion.
4. Insert the spinal needle perpendicular to the back at the level of L3-L4 or L4-L5 interspace. The needle should be inserted slightly cephalad, approximately at a 15° angle (Figure 2).
5. With the patient in a lateral recumbent position, keep the bevel of the cutting spinal needle up toward the ceiling. In the seated position, keep the bevel to either side as

this allows theoretical spreading rather than cutting of dural tissues. Use of a pencil-point spinal needle obviates the need for orientation of the needle tip.

6. Advance the needle incrementally while periodically removing the stylet. At times, a "pop" or a sudden decrease in resistance is noted when the dura is punctured. Frequent removal of the stylet while advancing the needle will reveal fluid if the dural layer is unknowingly crossed.
7. If bone is encountered, palpate the surrounding structures to ensure midline positioning. Withdraw the needle almost to the skin and re-angle slightly more cephalad as this may help avoid hitting the inferior spinous process. If unsuccessful, consider a different interspace, lateral or cisternal approach (not described here), ultrasound or fluoroscopic guidance.
8. With the patient in the lateral recumbent position, attach the manometer to obtain opening pressures. The patient must slowly straighten the legs and torso in order to obtain accurate measurements.
9. Take 1 to 4 mL of CSF per tube, enough for desired studies.
10. Re-insert the stylet and remove the spinal needle.
11. Clean and dress area (band-aid sufficient).

Complications

- Cerebellar herniation
- Pain – local or referred
- Bleeding
- Spinal epidural or subdural hematoma
- Infection
- Subarachnoid epidermal cyst formation
- CSF leakage
- Headache

Interpretation

- Interpretation of findings is based on whether the procedure is performed for diagnostic or therapeutic reasons. For diagnostic purposes, interpretation of findings including opening pressure will be heavily dependent on pretest probability of a certain pathology being present.
- See Table 1 and 2 for interpretation of opening pressure as well as CSF findings with workup for infectious purposes.
- Diagnosis of subarachnoid hemorrhage
  - Xanthochromia (yellowing of supernatant)
  - Serial cell counts showing no clearing of RBC's in the CSF

Table 1. Interpretation of opening pressure

< 70 mm H_2O	Improper needle placement (meningeal obstruction), CSF leak, barbiturate intoxication
70-180 mm H_2O	Normal
> 200 mm H_2O	Increased ICP: cerebral edema, increased CSF, inflammation, tumor

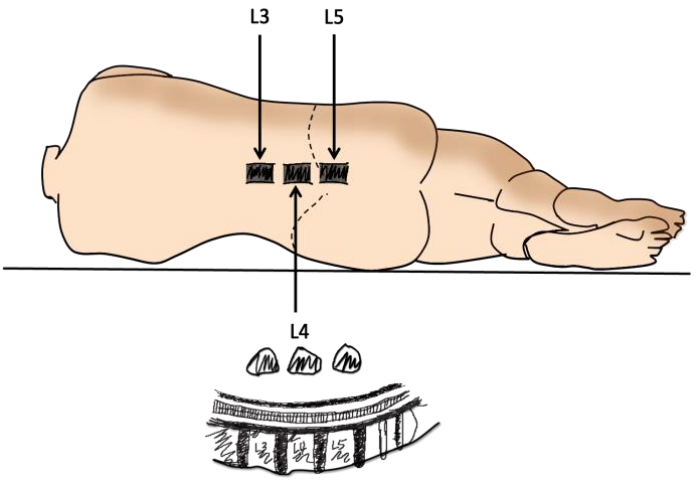


Figure 1. Lateral positioning.

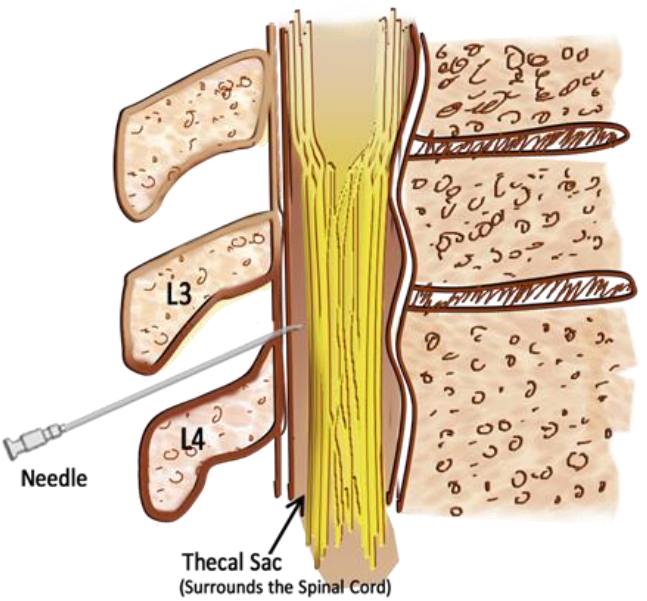


Figure 2. Needle insertion.

Table 2. CSF findings in infectious meningitis

	Bacterial	Viral	Fungal	TB
Opening Pressure	Elevated	Normal	Variable	Variable
WBC count	>1000 / mm3	<1000 / mm3	Variable	Variable
WBC Differential	Granulocytic predominance	Lymphocytic predominance	Lymphocytic predominance	Lymphocytic predominance
Protein	>200 mg/dL	<200 mg/dL	>200 mg/dL	>200 mg/dL
Glucose	Low	Normal	Low	Low

References

http://aianpramadhan.blogspot.com/2011/11/prosedur-lumbal-pungsi.html

http://www.headache-treatment-options.com/pseudotumor-cerebri.html

http://www.mayoclinic.com/health/medical/IM01849

Matata C, Michael B, Garner V, Solomon T. Lumbar puncture: diagnosing acute central nervous system infections. *Nurs. Stand*. 2012; 27(8):49–56; quiz 58.

Sempere AP, Berenguer-Ruiz L, Lezcano-Rodas M, Mira-Berenguer F, Waez M. [Lumbar puncture: its indications, contraindications, complications and technique]. *Rev. Neurol*. 2007; 45(7):433–436.

Straus SE, Thorpe KE, Holroyd-Leduc J. How do I perform a lumbar puncture and analyze the results to diagnose bacterial meningitis? *JAMA*. 2006; 296(16):2012–2022.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 18

Noninvasive Positive Pressure Ventilation

Renato Blanco, MD[1], Sri Sujanthy Rajaram, MD[2] and Archana Mishra, MD[3]
[1]St. Francis Medical Center, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[3]State University of New York at Buffalo, Buffalo, NY, US

Introduction

Noninvasive Positive Pressure Ventilation is a means of ventilatory support that can be used in the intensive care unit (ICU) setting as well as outside the ICU. The benefit of noninvasive ventilation is that it provides ventilatory support for patients without the use of an endotracheal tube or tracheostomy. The two types most often used are bilevel positive airway pressure (BPAP) and continuous positive airway pressure (CPAP). BPAP provides support by applying both inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). By decreasing the inspiratory effort through IPAP, and recruiting atelectatic alveoli through EPAP, a patient's work of breathing can be decreased. Continuous positive airway pressure provides support by supplying continuous positive pressure, thus recruiting alveoli and allowing breathing through more compliant portions of the lung. One important prerequisite for CPAP is that the patient must be able to breathe spontaneously. Another point to be aware of is that hemodynamic instability may result in patients with borderline low blood pressure as CPAP reduces venous return. However by reducing left ventricular transmural pressure it increases cardiac output.

Indication

- Chronic Obstructive Pulmonary Disease Exacerbation
- Cardiogenic Pulmonary Edema
- Hypoxemic Respiratory Failure
- Post-extubation
- Neuromuscular disorders
- Obstructive Sleep Apnea
- Obesity Hypoventilation Syndrome

Contraindication

- Cardiac Arrest or unstable cardiorespiratory status
- Respiratory Arrest
- Facial Trauma/Deformity
- Burns involving the face
- Severe GI bleed
- Severely Impaired Consciousness (Encephalopathy)
- High Aspiration Risk with inability to protect the airways
- Inability to clear secretions with weak cough reflex and/or excessive secretions
- Uncooperative patients

Preparation

Equipment

- Ventilator
  - Standard or Portable
  - BPAP is a pressure limited ventilation and Pressure Support is the difference between the IPAP and EPAP. The ventilator delivers air at this pressure each time the patient initiates a breath. A back up respiratory rate can be set to deliver a set number of breaths per minute.
  - Volume limited ventilation can also be used non-invasively with the interphase being the conduit between the patient and the ventilator. The ventilator can be set in assist control mode but higher tidal volume needs to be set to compensate for leaks.
  - Proportional assist ventilation is a mode in which the ventilator generates pressure in proportion to the patient's effort. The applied pressure is designed to overcome the elastic and resistance loads in proportion to the patient's volume and flow of breathing.
- Interfaces
  - Full Face Mask/Oronasal Mask

- Advantages- decreased air leaks
- Disadvantages- claustrophobia, difficult to monitor for aspiration

- Nasal mask/Nasal Prongs
  - Advantages- comfort, ability to eat and speak
  - Disadvantages- increased air flow resistance, air leaks

Figure 1. Full Face Mask.

Procedure

- Assure that patient is in appropriately monitored location
- Position patient in bed or chair at angle >30°
- Choose ventilator and Interface
- Apply interface and assure proper fitting (minimize strap tension by allowing one to two fingers under strap)
- Connect interface to ventilator
- Initiate with low pressure settings (ex: IPAP/EPAP: 10/5, 12/6)
- Set backup respiratory rate 12-14
- Monitor clinically and titrate pressures gradually to alleviate dyspnea, maintain adequate tidal volumes, and decrease respiratory rate
- Titrate FiO2 to maintain saturation >90
- Eliminate air leaks, ventilator asynchrony
- Reassure and encourage patient to improve compliance
- Consider mild sedation for agitation
- May add humidifier to reduce nasal congestion and dryness
- Check blood gas 1-2 hours to assess response
- Adjust settings accordingly

- It is important to closely monitor the patient's clinical status, vital signs, and blood gases while on noninvasive ventilation therapy. If there is lack of improvement, the need for intubation needs to be considered in a timely fashion.

Complications

- Local ulceration and pressure necrosis at the application site of masks or straps
- Sinus Pain
- Eye Irritation
- Gastric Distention
- Preload reduction and hypotension rarely

References

Diaz, O; Iglesia, R; Ferrer, M; et al. Effects of noninvasive ventilation on pulmonary gas exchange and hemodynamics during acute hypercapnic exacerbations of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*, 1997, 156, 1840.

Keenan, SP; Sinuff, T; Cook, DJ; Hill, NS. Does noninvasive positive pressure ventilation improve outcome in acute hypoxemic respiratory failure? A systematic review. *Crit Care Med*, 2004, 32, 2516.

Liesching, T; Kwok, H; Hill, NS. Acute applications of noninvasive positive pressure ventilation. *Chest*, 2003, 124, 699.

Masip, J; Roque, M; Sánchez, B; et al. Noninvasive ventilation in acute cardiogenic pulmonary edema: systematic review and meta-analysis. *JAMA*, 2005, 294, 3124.

Soo, Hoo, GW; Santiago, S; Williams, AJ. Nasal mechanical ventilation for hypercapnic respiratory failure in chronic obstructive pulmonary disease: determinants of success and failure. *Crit Care Med*, 1994, 22, 1253.

Soroksky, A; Stav, D; Shpirer, I. A pilot prospective, randomized, placebo-controlled trial of bilevel positive airway pressure in acute asthmatic attack. *Chest*, 2003, 123, 1018.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 19

Paracentesis

Sachin Mohan, MD[2], Carol H. Choe, MD[2*]
and Sri Sujanthy Rajaram, MD, MPH[1]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US

Paracentesis is a method of removing fluid from the abdomen, performed for both diagnostic and therapeutic purposes.

Indications

- Determine etiology of new onset ascites
- Exclude infection (spontaneous bacterial peritonitis)
- Assess worsening clinical status of a patient with ascites (fever, abdominal pain, abdominal tenderness, altered mental status, hepatic encephalopathy)
- Symptomatic relief of tense ascites

Contraindications

No absolute contraindications

Relative contraindications

- Acute abdomen that requires surgery

* Email: dr.carolchoe@gmail.com.

- Overlying infection/abdominal wall cellulitis at site of puncture
- Uncorrected bleeding diathesis (platelets < 25, INR > 3)
- Intra-abdominal adhesions
- Uncooperative patient
- Large volume paracentesis - relatively contraindicated in ARF and hepatorenal syndrome

Preparation

- Obtain informed consent from the patient or an appropriate surrogate
- Ultrasound guidance to determine appropriate site for catheter placement.
- Sterile gloves
- Chloraprep or iodine swabs
- 1% Lidocaine (10 mL vial)
- Prepackaged paracentesis tray
- 1 liter vacuum bottles (for therapeutic paracentesis)
- Band-aid
- For specimen collection:
  - Purple top tube: Cell count and differential
  - Red top tube: Albumin, LDH, protein if clinically indicated
  - Aerobic and anaerobic blood culture bottles
  - IV Albumin – 10 grams per liter of fluid removed (ONLY if taking off > 5 liters)

Procedure

1. Position: Most commonly done in the supine position with the patient slightly turned to the ipsilateral side of proposed needle insertion and head of bed raised to 45-60° (allows fluid to accumulate in the lower abdomen). Occasionally, "face-down" or prone position is favored for small fluid volumes where diagnostic paracentesis is essential for therapy and prognosis.
2. Site: An ultrasound of the abdomen helps estimate the best "pocket" for fluid aspiration. The distance from the skin to the fluid collection and to the midpoint of the fluid collection is determined, usually approximately 1 cm and 3 cm respectively.
   a. Bilateral lower quadrants, about 2-4 cm medially and caudally to the anterior superior iliac spine (ASIS), are possible sites for paracentesis. Left lower quadrant (LLQ) is preferred to the right as the abdominal wall is thinner and the fluid pocket is deeper on the left side.
   b. In current practice, the midline infraumbilical location (2 cm below the umbilicus) is seldom used. Though a relatively avascular site, the body wall is thicker here making the ascitic fluid more difficult to access.
   c. When using the lower quadrant, the ASIS is first identified and then a spot 3cm (two finger breadths) medial and rostral to it is marked (Figure 2a). As the ASIS

can be hard to identify in the morbidly obese patient, it is good practice to verify dullness to percussion and absence of a palpable spleen. Even with correct exam technique, ultrasound guidance should be used for improved patient safety.

3. After skin sterilization, anesthetize the skin and deeper tissues with 1% lidocaine.
4. Needle choice: For a *diagnostic paracentesis*, the narrowest needle possible is preferred to minimize complications. Metal needles are believed to be safest. For a *therapeutic paracentesis*, a small incision (using the supplied scalpel) is made and the pigtail catheter needle setup is then advanced through the incision (but with increased chances of a post-paracentesis leak).
5. With the non-dominant hand, the skin is retracted prior to inserting and advancing the needle. Theoretically, the z-track maneuver reduces the risk of fluid leak as holes in skin and peritoneum will not line up after the needle is withdrawn (Figure 2b)
6. Applying negative pressure to the syringe, the needle is advanced into the peritoneum until fluid return is noted.
7. Once fluid collection is complete, the needle should be withdrawn in one smooth movement to minimize pain.

Complications

- Peritoneal fluid leak (5%)
- Infection (peritonitis)
- Perforated viscous (small or large bowel, stomach, bladder)
- Hemorrhage (< 0.2%)
- Abdominal wall hematoma
- Spontaneous hemoperitoneum – rare, secondary to mesenteric variceal bleeding after removal of a large amount of ascitic fluid (>4 L).
- Catheter laceration and loss in abdominal cavity
- Laceration of major blood vessel (aorta, mesenteric artery, iliac artery)
- Post-paracentesis circulatory dysfunction (PCD) – defined as a 50% increase in renin activity is considered objective evidence of hypovolemia. It is associated with hypotension, hyponatremia, renal failure, hepatorenal syndrome and death in severe cases

Interpretation

- Color
  - Bloody fluid: HCC (30% of patients), malignancy, TB (rare)
  - Milky: cirrhosis, malignancy, lymphoma, TB
  - Turbid: peritonitis, bowel perforation or infection, or ischemic bowel
- Cell count and differential
  - Ascitic neutrophil count >250 cells/μL is consistent with SBP
  - Lymphocytic predominance is concerning for tuberculous ascites

- Cultures
- Chemistry
  - Total protein <1 g/dL is susceptible to SBP
  - Glucose is often lower in infection (gut perforation) and malignancy
  - LDH is elevated in infection and malignancy
  - Amylase (fluid/serum ratio > 0.4) is concerning for pancreatitis and gut perforation
  - Triglyceride level should be measured when milky fluid is obtained. A triglyceride concentration >200mg/dL is consistent with chylous ascites.
  - Bilirubin level is obtained when peritoneal fluid is very brown. A level >6mg/dL is suggestive of bowel / biliary perforation
- Cytology should be obtained if there is concern for peritoneal carcinomatosis. There is increased sensitivity with increased volume (at least 50mL)

ALWAYS ensure you have a recent albumin count to calculate the SAAG (Serum Ascites Albumin Gradient) (Table 1)

Table 1. Serum-to-ascites albumin gradient: (Runyon et al. 1992)

Portal Hypertension related ≥1.1g/dL	Non-portal Hypertension related <1.1g/dL
Cirrhosis	Peritoneal carcinomatosis
Alcoholic hepatitis	Peritoneal TB
CHF	Pancreatitis
Massive hepatic metastases	Serositis
Vascular occlusion	Nephrotic syndrome
Fatty liver disease of pregnancy	Bowel obstruction / infarction / perforation
Myxedema	

Considerations for Therapeutic Paracentesis

- How much to remove? Large volume paracentesis (LVP) – defined as >5 liters.
  - For tense ascites, remove enough fluid to relieve the intra-abdominal pressure (IAP) and minimize chances of an ascitic fluid leak.
  - If diuretic-responsiveness is unknown, removal of approximately 5 liters is enough to reduce IAP.
  - In known cases of refractory ascites, the strategy is to remove as much fluid as possible to extend the time interval to the next paracentesis.
- Vacuum bottles should be used to speed removal of fluid.
- As the fluid is drained, the bowel and omentum are more likely to occlude the needle hole(s) and slow or stop the flow of fluid. If flow decreases, slowly and gently reposition the patient to pool fluid at the needle site.

- Need for colloid replacement after LVPs remains controversial. The rationale for replacement is to avoid intravascular shift, prevent hypovolemia and consequent post-paracentesis circulatory dysfunction
  - A meta-analysis showed survival benefit with albumin infusion
  - Current practice is to give albumin (6 to 8g for each liter of fluid removed) during LVP to prevent morbidity. In the U.S., the practice is to give 25mL of 25% albumin for every 2L of ascitic fluid evacuated.

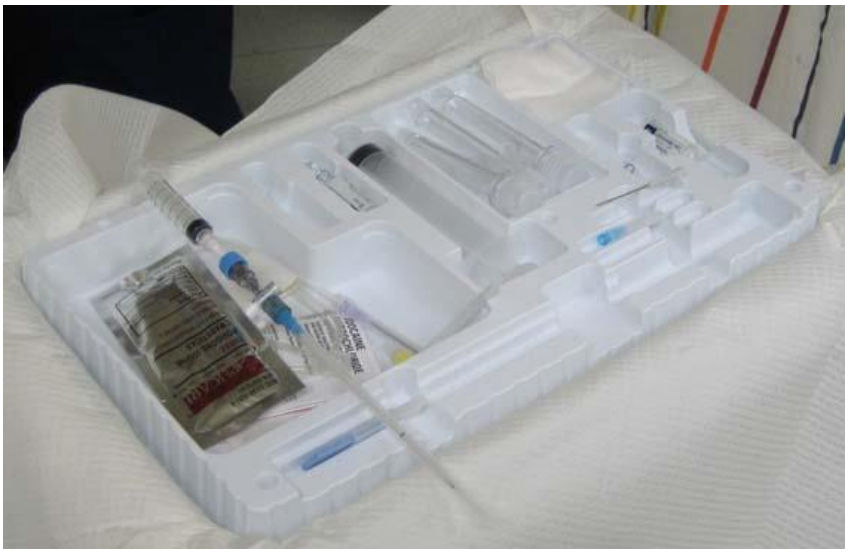
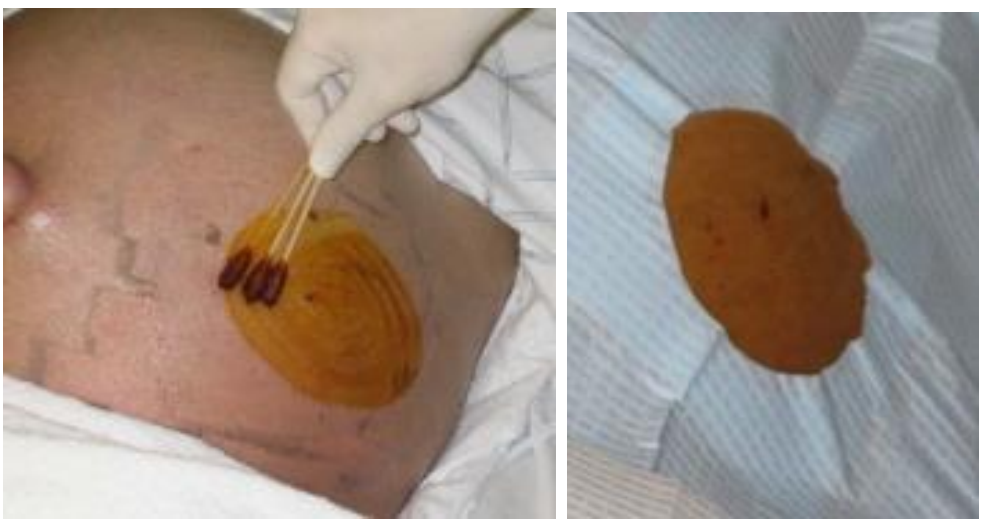


Figure 1. Typical paracentesis tray.



(a) (b)

Figure 2. (a) Prepping and (b) draping the desired location.

References

Alessandria C, Elia C, Mezzabotta L, et al. Prevention of paracentesis-induced circulatory dysfunction in cirrhosis: standard vs half albumin doses. A prospective, randomized, unblinded pilot study. *Dig Liver Dis*. 2011;43(11):881–886.

Bernardi M, Caraceni P, Navickis RJ, Wilkes MM. Albumin infusion in patients undergoing large-volume paracentesis: a meta-analysis of randomized trials. *Hepatology*. 2012;55(4):1172–1181.

Grabau CM, Crago SF, Hoff LK, et al. Performance standards for therapeutic abdominal paracentesis. *Hepatology*. 2004;40(2):484–488.

http://emedicine.medscape.com/article/80944-overview#a17.

Runyon BA, AASLD Practice Guidelines Committee. Management of adult patients with ascites due to cirrhosis: an update. *Hepatology*. 2009;49(6):2087–2107.

Runyon BA, Montano AA, Akriviadis EA, et al. The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. *Ann. Intern. Med.* 1992;117(3):215–220.

Runyon BA. Diagnostic and therapeutic abdominal paracentesis. In: *UpToDate*, Chopra S (ed), UpToDate, Waltham, MA.

Runyon BA. Evaluation of patients with ascites. In: *UpToDate*, Lindor KD (ed), UpToDate, Waltham, MA.

Runyon MS, Marx JA. Peritoneal Procedures. In: Roberts JR, Hedges JR, eds. *Clinical Procedures in Emergency Medicine*. 4th ed. Philadelphia, PA: Saunders. On-line version available at MD Consult Books on Healthlinks (http://healthlinks. washington.edu).

Sakai H, Sheer TA, Mendler MH, Runyon BA. Choosing the location for non-image guided abdominal paracentesis. *Liver Int.* 2005;25(5):984–986.

Thomsen TW, Shaffer RW, White B, Setnik GS. Videos in clinical medicine. Paracentesis. *N. Engl. J. Med.* 2006;355(19):e21.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 20

Percutaneous Tracheostomy

Edward Peter Mossop, MD and Adel Bassily-Marcus, MD*
Division of Critical Care, Department of Surgery
Mount Sinai School of Medicine, New York, NY, US

Introduction

Tracheostomy is frequently performed in the intensive care unit (ICU) to facilitate weaning from mechanical ventilation, reduce anatomical deadspace, avoid laryngeal injury and aid in management of tracheobronchial and pulmonary secretions.

Tracheostomy is one of the earliest recorded surgical procedures, performed primarily to relieve upper airway obstruction, with descriptions portrayed on Egyptian tablets dating back to 3600 BC [1].

Chevalier Jackson developed a standard open operative technique in 1909 [2] for patients requiring long-term mechanical ventilation, and is still used to this day. However, open tracheostomy requires skilled open neck dissection, usually in the operating room, and can be associated with hemorrhage, subglottic stenosis and cosmetic deformity [3-5].

Several percutaneous techniques have been described over the years [6, 7], however it was Pasquale Ciaglia in 1985 that described the method of bedside percutaneous dilational tracheostomy (PDT) over a Seldinger guidewire using increasing sizes of hydrophilic coated dilators between the tracheal rings [8].

This technique of tracheostomy has evolved to include ultrasound and fiberoptic bronchoscopy as adjuncts to aid visualization and improve safety. The use of sequential dilators has been replaced by a single, tapered and curved dilator [9] to reduce point pressure on the posterior trachea and limit procedural time and tidal volume loss from changing dilators [10].

* Corresponding author: E.mail: adel.bassily-marcus@mountsinai.org.

Variations of the percutaneous approach include the controlled rotating dilation method (PercuTwist) [11], the Griggs' forceps dilational tracheostomy [12] and the balloon dilation percutaneous dilational tracheostomy technique [13, 14].

PDT involves limited tissue dissection and therefore less bleeding, fewer wound complications such as infection and less cosmetic deformity when compared to open tracheostomy [15]. A meta-analysis comparison of PDT to open surgical technique, demonstated a trend towards greater feasibility, more favorable cost effectiveness and fewer complications, with percutaneous techniques [16].

However the main advantage of PDT is the facility to perform the procedure early at the bedside in the ICU thus avoiding the transfer of unstable patients to the operating room. Numerous studies have shown that skilled operators can perform bedside PDT with outcomes as good as those associated with open surgical tracheostomy [17-20].

PDT has been increasingly performed in the ICU since 1985, and has gained wide acceptance across all ICUs having been shown to be safe and cost effective even in high-risk patients. Recently, it has been considered the new gold standard for tracheostomies in critically ill patients in ICUs worldwide [21, 22]. For selected patients however, open surgical technique remains the procedure of choice where PDT is either technically difficult or carries an increased risk for major complications.

Indications

The majority of PDT performed in ICUs are done primarily to provide airway access for prolonged mechanical ventilation. It facilitates mechanical ventilation without the need for sedation, provides a more secure airway, enables physical therapy and intermittent weaning trials, facilitates access for secretion removal, improves patient comfort, and promotes progression of care in and outside the intensive care unit.

Timing of tracheostomy has been controversial with no clear benefit demonstrated from performing tracheostomy early (within 7 days) vs late (14 days) in the ICU [23-26] including patients with stroke [27].

Contraindication

Strong Contraindications: While the majority of tracheostomies performed in critically ill patients can be done percutaneously, open surgical tracheostomy remains the alternative choice for selected patients where the percutaneous approach imposes a significant risk, or is technically difficult due to challenging anatomy. Examples include; very low cricoid, high innominate artery, large nodular goiter with thick isthmus, prior neck dissection, and when the trachea has been displaced due to tumor, scar, goiter, kypho-scoliosis, cervical irradiation and neck trauma.

Relative contraindications: These include; unstable cervical spine injury (preventing neck extension) and difficult landmarks/anatomy for example in the obese, or those with a short neck (cricoid <3cm above the sternal notch), an aberrant innominate artery and prior neck surgery. Known or suspected difficult intubation, which can lead to difficulties

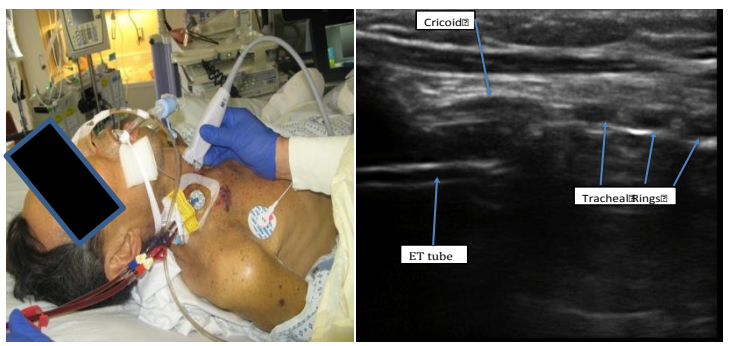
managing the airway during the procedure, should also be taken into consideration. High oxygen and positive end expiratory pressure (PEEP) requirements may impose a difficulty during bronchoscopy or during dilation due to loss of PEEP and hypoventilation.

PDT has been shown to be safe in patients with an increased bleeding risk such as low platelets and elevated INR [28].

Preparation

PDT is a planned non-emergency procedure therefore all appropriate staff and equipment should be available and the procedure should be performed during normal working hours when support staff and facilities are readily available.

An experienced clinician must perform a thorough clinical examination of the anterior neck anatomy as well as an assessment of the airway to identify potential difficulties and contraindications. Perioperative ultrasound of the neck may help identify structures at risk for hemorrhage, such as aberrant blood vessels [29-31] (Figure 1).



A B

Figure 1. The ultrasound probe is placed on the neck in the longitudinal plane (A) and anatomy is identified on ultrasound (B).

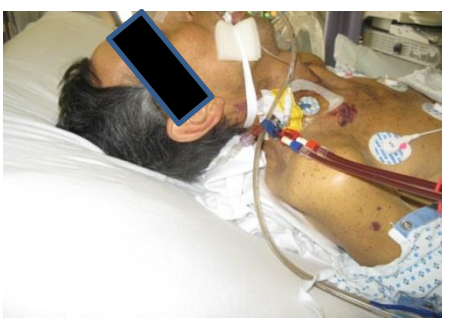


Figure 2. supine position and neck extended to open the tracheal interspaces with a supporting towel placed between the scapulae.

Tube feeds and any anticoagulation should be held similar to other operative procedures.

The patient should be placed on continuous monitoring of heart rate, oxygen saturation, and blood pressure. The ventilator should be adjusted to provide 100% oxygen and a higher respiratory rate in anticipation for loss of PEEP and hypoventilation. Emergent airway equipment including an intubation kit, bronchoscope and a tray for open tracheostomy should be at the bedside. For bronchoscopic guidance, a second skilled operator should be preparing and operating the bronchoscope and the necessary equipments.

Procedure

PDT is performed under IV sedation and analgesia with suitable agents. Neuromuscular blockers are often not needed. The patient is placed in the supine position with the neck hyperextended to open the tracheal interspaces with a suitable supporting towel placed between the scapulae (Figure 2) Although airway procedures are semi sterile, sterile precautions are always taken. The patient must be fully draped with sterile sheets and the physician performing the PDT must use a cap, mask, gloves, sterile gown and a protective eye shield. All staff assisting should wear personal protective equipment (Figure 3). The PDT kit should be opened and laid out in an organized fashion (Figure 4). The tracheostomy tube should be inspected, prepared and the balloon tested.

The tracheal rings, cricoid cartilage and suprasternal notch should be palpated again under sterile precautions. Local anesthesia with epinephrine is infused into the surgical site (Figure 5). A 2cm horizontal skin incision is then made over the 2nd and 3rd tracheal rings, approximately halfway between the palpable cricoid and the suprasternal notch. The wound is then bluntly dissected with a hemostat through the subcutaneous fascia (Figure 6).

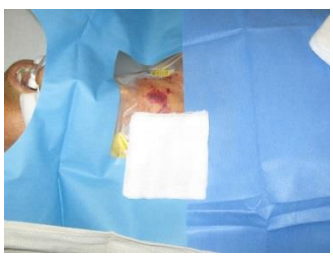


Figure 3. patient is prepped and draped using standard sterile technique with the proposed tracheostomy site exposed.

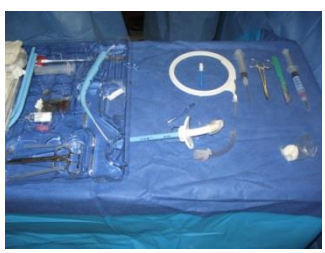


Figure 4. Standard Ciaglia percutanous tracheostomy kit.

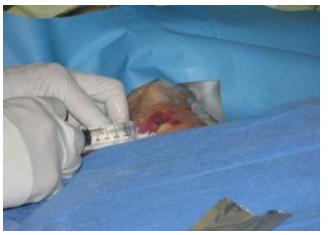


Figure 5. Lidociane 1% with epinephrine is infiltrated to provide local anesthesia.

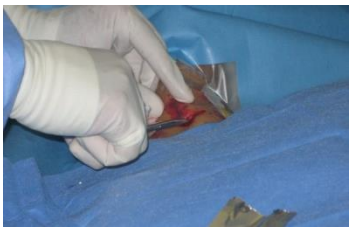


Figure 6. After 2cm horizontal incision is made over the 2nd and 3rd tracheal rings, the area is bluntly dissected with a hemostat.

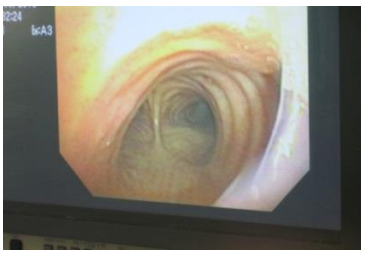
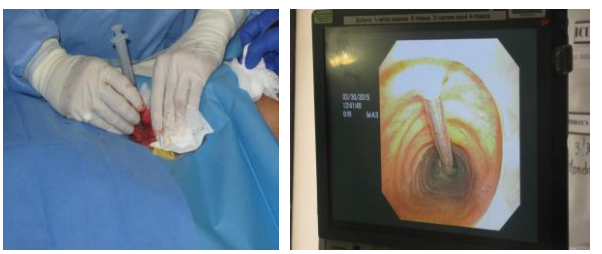


Figure 7. Bronchoscope is inserted so that the distal part of the endotracheal tube (ETT) is still visible. The ETT is then slowly withdrawn to a position above the 1st tracheal ring.

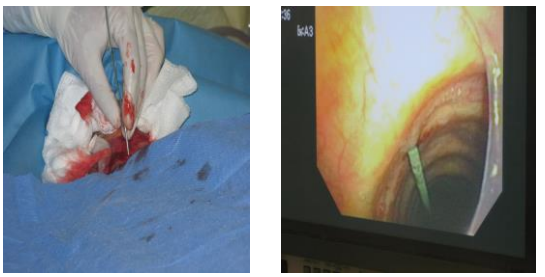


A B

Figure 8. The skin is punctured (A) and the tracheal is cannulated with an introducer needle under direct visualization (B).

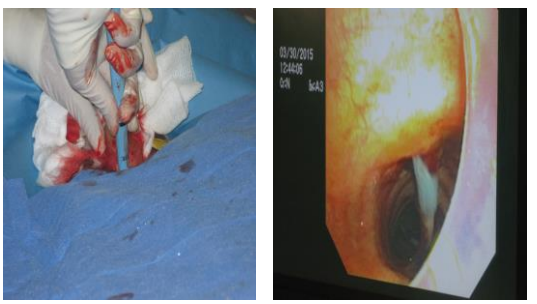
An appropriately skilled second operator then inserts a fiberoptic bronchoscope into the endotracheal tube (ETT) and the tube is withdrawn carefully into a position above the first tracheal ring ensuring the cuff remains at or just below the vocal cords. The bronchoscope is inserted in such a way that the distal tip of the ETT remains visible on the screen at all times

(Figure 7). The trachea is then cannulated through the skin incision with the introducer needle under direct visualization via the bronchoscope without impaling the ETT (Figure 8).



A B

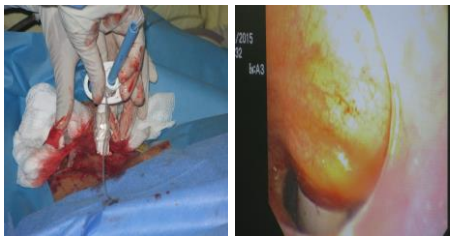
Figure 9. A guidewire is advanced through the needle (A) towards the carina under direct visualization (B) and then the needle is withdrawn.



A B

Figure 10. Single beveled curved dilator is inserted over the guidewire (A), under direct visualization (B).

A J tipped guidewire is advanced through the needle towards the carina, under direct visualization, and the needle is withdrawn (Figure 9). A 14 French stiff dilator is then passed over the wire to create a tracheostoma and then removed. A single beveled curved dilator is then advanced over the guide wire through the incision, under direct visualization, dilating the tracheostoma (Figure 10) till the mark on the dilator reaches the skin level. Once the trachestoma has been dilated to the appropriate size, a specialty tracheostomy tube is introduced into the trachea over the same guide wire using the inner part of the dilator as an obturator. The position is confirmed via the bronchoscope and the obturator and guide wire are removed (Figure 11). The bronchoscope is then passed directly through the tracheotomy to confirm placement (Figure 12) in the airway and confirming no false passage. Once the position is confirmed, the ballon is inflated and an inner cannula and swivel adaptor is inserted. The ETT is then removed and the patient is connected to the ventilator. The physician should then monitor the exhaled tidal volume to detect any air leak. The tracheostomy tube is then sutured in place with 4 interrupted sutures using 2/0 polypropyelene sutures.



A B

Figure 11. Tracheostomy tube is inserted into the trachea over the guidewire (A) and confirmed with direct visualization (B).

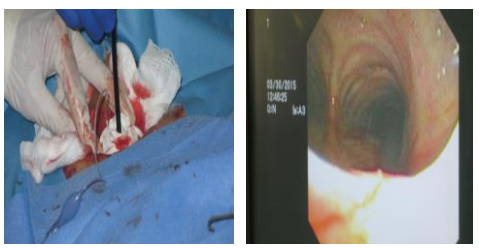


Figure 12. The bronchoscope is then passed directly through the new tracheostomy tube (A) to confirm placement (B).

A chest x-ray should be performed immediately following the procedure to confirm the position of the tracheostomy tube and to detect any potential complications such as pneumothorax, subcutaneous emphysema, lung collapse and significant aspiration. Every time the patient is repositioned, care must be taken to avoid dislodgement of the tube.

Complications

Manipulation of the airway while sacrificing a functional ventilation system constitutes a high-risk endeavor.

Major perioperative complications include; pneumothorax, pneumomediastinum, accidental loss of the airway, false passage of the wire, dilator and tracheostomy tube, posterior tracheal wall injury, bleeding, aspiration and death [32, 37]. However these complications occur at a very low rate ranging from 0.38 % [22] to 7% [33, 34].

When correctly placed, tracheostomies will traverse the thyroid isthmus in approximately 30% [35] which poses an increased bleeding risk, however bleeding with PDT is lower than with surgical tracheostomy because the procedure is largely dilational and the tightly fitting tracheotomy tube may function to tamponade the bleeding vessels [3, 4, 36].

If a newly inserted (<1 week) tracheostomy tube dislodges, orotracheal intubation is the safest method to reestablish a patent airway [38]. No attempts should be made to re-advance the dislodged tracheostomy tube through a newly formed tract.

Longer term postoperative complications are rare and can include bleeding and tracheal stenosis. The incidence compares favorably with that of surgical tracheostomy [39]. Any

arterial bleeding from or around the tracheostomy site several days or months post insertion should be considered to be a tracheal-innominate artery fistula, which requires immediate surgical attention, however this is rare occurring in around 0.3% of cases.

Ultrasound

Ultrasound guided PDT techniques have been well described [29, 40, 41] and its use has numerous advantages.

Ultrasound prior to the procedure at the intended site can help define the anatomy of the trachea, allow accurate assessment of depth from the skin surface and help identify large superficial and deep vessels that were neither visible nor palpable [29] (Figure 1). As a screening tool, ultrasonography helps stratify candidacy for percutaneous approach vs open, decreasing the procedure's risks. [30]

Bronchoscopy

Bronchoscopy assists with guidance of the needle used to enter the airway and confirms the position of the wire, dilator and tracheostomy in the correct position within the airway (Figures 7, 8, 9, 10 and 11).

Several authors have reported the safety of PDT without bronchoscopy when performed by experienced hands [22, 42]. However, others have cautioned against removing imaging guidance, particularly bronchoscopy, which exposes patients to the unnecessary risk of serious complications [43].

In a recent retrospective cohort study comparing ultrasound-guided PDT to bronchoscopy-guided PDT, there were similar low complication rates and clinical outcomes. However, the ultrasound guided procedure was relatively shorter in duration [44].

References

[1] Pahor, AL. Ear, nose and throat in Ancient Egypt. *J Laryngol Otol*, 1992. 106(9), 773-9.

[2] Jackson, C. *Tracheotomy*, 1909, St. Louis?: s.n. 8 p.

[3] Stoeckli, SJ., Breitbach, T; Schmid, S. A clinical and histologic comparison of percutaneous dilational versus conventional surgical tracheostomy. *Laryngoscope*, 1997. 107(12 Pt 1), 1643-6.

[4] Walz, MK; et al. Percutaneous dilatational tracheostomy--early results and long-term outcome of 326 critically ill patients. *Intensive Care Med*, 1998. 24(7), 685-90.

[5] Antonelli, M; et al. Percutaneous translaryngeal versus surgical tracheostomy: A randomized trial with 1-yr double-blind follow-up. *Crit Care Med*, 2005. 33(5), 1015-20.

[6] Sheldon, C; Pudenz, R; Tichy, F. Percutaneous tracheostomy. *JAMA*, 1957. 165, 2068-2070.

[7] Toy, FJ; Weinstein, JD. A percutaneous tracheostomy device. *Surgery*, 1969. 65(2), 384-387.

[8] Ciaglia, P; Firsching, R; Syniec, C. Elective percutaneous dilatational tracheostomy. A new simple bedside procedure; preliminary report. *Chest*, 1985. 87(6), 715-9.

[9] Byhahn, C; et al. [Ciaglia blue rhino: a modified technique for percutaneous dilatation tracheostomy. Technique and early clinical results]. *Anaesthesist*, 2000. 49(3), 202-6.

[10] Johnson, JL; et al. Percutaneous dilational tracheostomy: a comparison of single- versus multiple-dilator techniques. *Crit Care Med*, 2001. 29(6), 1251-4.

[11] Westphal, K; et al. PercuTwist: a new single-dilator technique for percutaneous tracheostomy. *Anesth Analg*, 2003. 96(1), 229-32, table of contents.

[12] Griggs, WM; et al. A simple percutaneous tracheostomy technique. *Surg Gynecol Obstet*, 1990. 170(6), 543-5.

[13] Gromann, TW; Birkelbach, O; Hetzer, R. Balloon dilatational tracheostomy: initial experience with the Ciaglia Blue Dolphin method. *Anesth Analg*, 2009. 108(6), 1862-6.

[14] Zgoda, MA; Berger, R. Balloon-facilitated percutaneous dilational tracheostomy tube placement: preliminary report of a novel technique. *Chest*, 2005. 128(5), 3688-90.

[15] Silvester, W; et al. Percutaneous versus surgical tracheostomy: A randomized controlled study with long-term follow-up. *Crit Care Med*, 2006. 34(8), 2145-52.

[16] Higgins, KM; Punthakee, X. Meta-analysis comparison of open versus percutaneous tracheostomy. *Laryngoscope*, 2007. 117(3), 447-54.

[17] Freeman, BD; et al. A prospective, randomized study comparing percutaneous with surgical tracheostomy in critically ill patients. *Crit Care Med*, 2001. 29(5), 926-30.

[18] Westphal, K; et al. Percutaneous tracheostomy: a clinical comparison of dilatational (Ciaglia) and translaryngeal (Fantoni) techniques. *Anesth Analg*, 1999. 89(4), 938-43.

[19] Angel, LF; Simpson, CB. Comparison of surgical and percutaneous dilational tracheostomy. *Clin Chest Med*, 2003. 24(3), 423-9.

[20] Melloni, G; et al. Surgical tracheostomy versus percutaneous dilatational tracheostomy. A prospective-randomized study with long-term follow-up. *J Cardiovasc Surg (Torino)*, 2002. 43(1), 113-21.

[21] Kornblith, LZ; et al. One thousand bedside percutaneous tracheostomies in the surgical intensive care unit: time to change the gold standard. *J Am Coll Surg*, 2011. 212(2), 163-70.

[22] Dennis, BM; et al. Safety of bedside percutaneous tracheostomy in the critically ill: evaluation of more than 3,000 procedures. *J Am Coll Surg*, 2013. 216(4), 858-65, discussion 865-7.

[23] Terragni, PP; et al. Early vs late tracheotomy for prevention of pneumonia in mechanically ventilated adult ICU patients: a randomized controlled trial. *JAMA*, 2010. 303(15), 1483-9.

[24] Young, D; et al. Effect of early vs late tracheostomy placement on survival in patients receiving mechanical ventilation: the TracMan randomized trial. *JAMA*, 2013. 309(20), 2121-9.

[25] Andriolo, BN; et al. Early versus late tracheostomy for critically ill patients. *Cochrane Database Syst Rev*, 2015. 1, CD007271.

[26] Terragni, P; et al. Tracheostomy in mechanical ventilation. Semin Respir *Crit Care Med*, 2014. 35(4), 482-91.
[27] Lee, YC; et al. Comparison of Complications in Stroke Subjects Undergoing Early Versus Standard Tracheostomy. *Respir Care*, 2015.
[28] Rosseland, LA; Laake, JH; Stubhaug, A. Percutaneous dilatational tracheotomy in intensive care unit patients with increased bleeding risk or obesity. A prospective analysis of 1000 procedures. *Acta Anaesthesiol Scand*, 2011. 55(7), 835-41.
[29] Hatfield, A; Bodenham, A. Portable ultrasonic scanning of the anterior neck before percutaneous dilatational tracheostomy. *Anaesthesia*, 1999. 54(7), 660-663.
[30] Bassily-Marcus, A; et al. IMproving safety of percutaneous tracheostomy using ultrasound screening. *Chest*, 2011. 140(4_MeetingAbstracts), 893A-893A.
[31] Farmery, A; Shlugman, D; Anslow, P. How high do the subclavian arteries ascend into the neck? A population study using magnetic resonance imaging. *British journal of anaesthesia*, 2003. 90(4), 452-456.
[32] Simon, M; et al. Death after percutaneous dilatational tracheostomy: a systematic review and analysis of risk factors. *Crit Care*, 2013. 17(5), R258.
[33] Berrouschot, J; et al. Perioperative complications of percutaneous dilational tracheostomy. *Laryngoscope*, 1997. 107(11 Pt 1), 1538-44.
[34] Kearney, PA; et al. A single-center 8-year experience with percutaneous dilational tracheostomy. *Ann Surg*, 2000. 231(5), 701-9.
[35] Dexter, TJ. A cadaver study appraising accuracy of blind placement of percutaneous tracheostomy. *Anaesthesia*, 1995. 50(10), 863-4.
[36] Gysin, C; et al. Percutaneous versus surgical tracheostomy: a double-blind randomized trial. *Ann Surg*, 1999. 230(5), 708-14.
[37] Lin, JC; Maley, RH Jr; Landreneau, RJ. Extensive posterior-lateral tracheal laceration complicating percutaneous dilational tracheostomy. *Ann Thorac Surg*, 2000. 70(4), 1194-6.
[38] Morris, LL; Whitmer, A; McIntosh, E. Tracheostomy care and complications in the intensive care unit. *Crit Care Nurse*, 2013. 33(5), 18-30.
[39] Fernandez, L; et al. Bedside percutaneous tracheostomy with bronchoscopic guidance in critically ill patients. *Arch Surg*, 1996. 131(2), 129-32.
[40] Kollig, E; et al. Ultrasound and bronchoscopic controlled percutaneous tracheostomy on trauma ICU. *Injury*, 2000. 31(9), 663-8.
[41] Sustic, A; et al. Ultrasound-guided percutaneous dilatational tracheostomy: a safe method to avoid cranial misplacement of the tracheostomy tube. *Intensive Care Med*, 2000. 26(9), 1379-81.
[42] La Scienya, MJ; et al. Percutaneous tracheostomy: to bronch or not to bronch—that is the question. Journal of Trauma and Acute Care *Surgery*, 2011. 71(6), 1553-1556.
[43] Corso, RM; Gambale, G. Percutaneous tracheostomy: let's play it safe. *J Trauma Acute Care Surg*, 2012. 73(3), 779-80, author reply 780.
[44] Gobatto, ALN; et al. Comparison between ultrasound-and bronchoscopy-guided percutaneous dilational tracheostomy in critically ill patients: A retrospective cohort study. *Journal of critical care*, 2014.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 21

Pulmonary Artery (Swan Ganz) Catheters

Nayan Desai, MD[2], Fiorella Nawar, MD[4], Mithil Gajera, MD[3], Munira Mehta, MD[5], and Sri Sujanthy Rajaram, MD, MPH[1*]

[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]Cooper Medical School of Rowan University, Camden, NJ, US
[3]Christiana Care hospital, Wilmington, DE, US
[4]Sparks Regional Medical Center, Fort Smith, AK, US
[5]Mercy Fitzgerald Hospital, Darby, PA, US

Introduction

The concept of right heart catheterization was first introduced by Dr Forrsmann in 1929. It was in 1970 that Dr HJ Swan and Dr William Ganz introduced the flow-directed balloon-tipped catheter that led to a paradigm shift in the way right heart catheterizations were performed at the bedside using intracardiac pressure tracings, without fluoroscopic guidance. Since then, the pulmonary artery catheter (PAC), also called a Swan-Ganz catheter, has been utilized in the management of intensive care unit (ICU) patients.

Indications

- Differentiation among various types of shock states
- Differentiation between high- vs low-pressure pulmonary edema

* Email: sujanty@gmail.com.

- Right ventricular failure
- Complex fluid management in the presence of impending renal failure
- Dynamic assessment of cardiac function in specific conditions
- Peri-operative management in cardiothoracic surgery patients
- Evaluation and assessment of reversibility of pulmonary hypertension (gold standard)

Contraindications

Absolute contraindications

- None

Relative contraindications

- Severe coagulopathy (correction of coagulopathy is recommended)
- Prosthetic right heart valve – catheter insertion may cause malfunction of the valve
- Pacemaker

Procedure

1. Place a single-lumen introducer sheath into the jugular vein, preferably on the right, using ultrasound guidance (see Chapter 10).
2. Open the PA catheter kit sterilely and remove only the thermistor port.
3. Sterilely insert the port into the cardiac output cable of the monitor. Allow the machine to calibrate itself.
4. Cover the connector and the proximal-most part of the cable with sterile towels to maintain aseptic technique as it rests on the sterile patient drapes.
5. Once calibrated, remove the rest of the catheter from the kit.
6. Flush all ports and attach one-way valves to their ends except the distal PA port
7. Connect the large round red and blue ports to their appropriate cables on the monitor. Again, cover these connections and cables appropriately.
8. Check the integrity of the balloon by inflating it with 0.5-1.5 mL of air using the supplied syringe. Once inflated, submerge the balloon in a cup of sterile water to assess for air leaks (bubbles). Then passively deflate the balloon by aspirating the air into the syringe.
9. Pass the catheter through the sterile protective sleeve, ensuring proper orientation of the sleeve.
10. Insert the deflated PAC with the sterile cover through the introducer sheath. The catheter has a slight curve which can be used to facilitate its direction into the pulmonary artery

11. Advance the catheter until it reaches the superior vena cava (SVC, approximately 15-20 cm from the right internal jugular vein). A venous pressure waveform will appear on the monitor.
12. Inflate the balloon with 1.5 mL of air and lock it in place.
13. Advance the catheter with the balloon inflated in order to allow the blood flow to guide it into the desired location. The catheter should pass from the SVC to the right atrium and then into right ventricle.
14. Once the right ventricle is reached (approximately 30 cm from the right IJ), a wide waveform with elevated pulmonary systolic pressures (15-30 mmHg) is visualized on the monitor. Continue to slowly advance the catheter.
15. If at any point the catheter is inserted to the appropriate estimated depth without visualizing corresponding changes in the waveform, deflate the balloon by aspirating air from the syringe, pull back the catheter to approximately 15 cm, re-inflate the balloon and reattempt to float the catheter into the pulmonary artery
16. When PA catheter reaches the pulmonary artery, the diastolic pressure wave will increase to 6-12 mmHg while the systolic pressure wave will remain unchanged.
17. Gently advancing the PAC further, the pulsatile PA pressure will transition to a venous pressure waveform at the level of the diastolic PA pressure. This is the pulmonary capillary wedge pressure (PCWP) or pulmonary artery occlusion pressure (PAOP).
18. Note the depth of PA catheter placement and deflate the balloon. After balloon deflation the PA pressure waveform will reappear.
19. Lock the PAC in place with the sterile sheath to prevent the catheter from migrating.
20. The balloon should always remain deflated except to intermittently check PCWP.

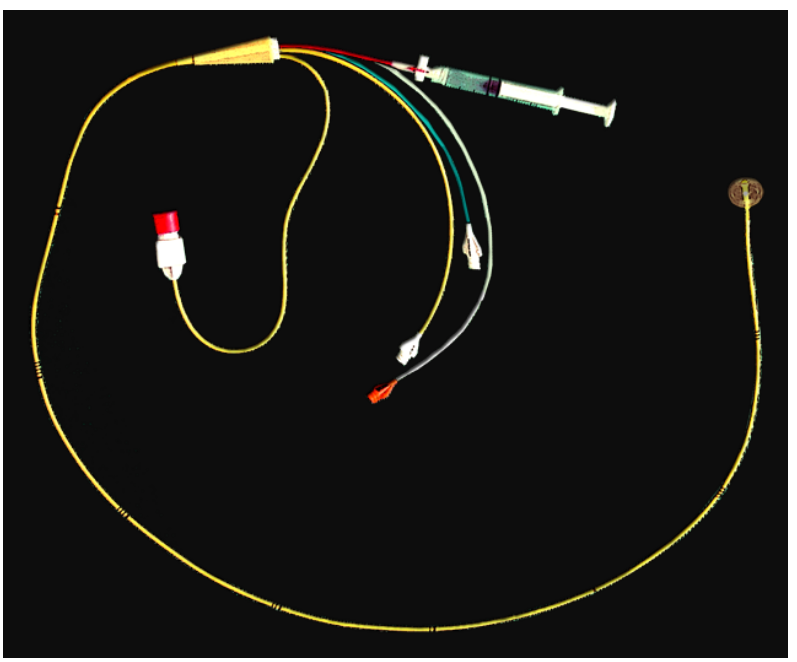


Figure 1. Pulmonary artery (Swan Ganz) Catheter with balloon inflated.

Interpretation

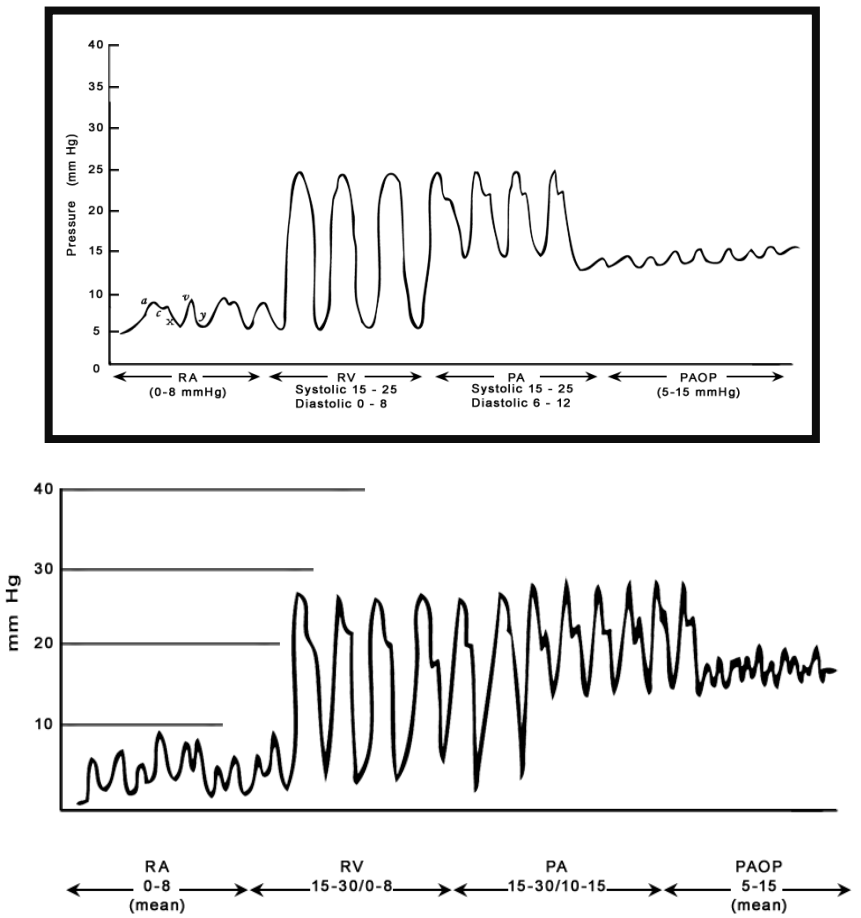


Figure 2. Waveforms seen with pulmonary artery catheter placement. a, atrial contraction; c, ventricular contraction; x, atrial relaxation; v, atrial venous filling; y, ventricular filling.

Table 1. Typical hemodynamic profile in various shock states

Shock hemodynamics						
	CO	SVR	PCWP	MAP	SVO_2	lactate
Cardiogenic	↓	↑	↑	↓	↓	↑
Obstructive (PE)	↓	↑	normal	↓	↓	↑
Obstructive (tamponade)	↓	↑	↑	↓	↓	↑
Distributive (early sepsis)	↑	↓	↓	↓ or N	↑	↑
Distributive (late sepsis)	↓	↑	normal	↓		↑
Hypovolemic	↓	↑	↓	↓	↓	↑

- Hypovolemic shock: decreased cardiac output and PCWP is seen as a result of decreased intravascular volume (ie: hemorrhage; hypovolemia secondary to vomiting, diarrhea)

- Cardiogenic shock: decreased cardiac output and increased PCWP as a result of cardiac pump failure.
- Distributive shock: characterized by low SVR and low PCWP secondary to peripheral vasodilation in early sepsis, whereas PCWP is normal late in the sepsis course.
- Obstructive shock (PE): presence of elevated right sided pressure (PA, RA and RV).
- Obstructive shock (tamponade): equalization and elevation of pressure of the RA, RV and PCWP.

Table 2. Typical pressures noted in shock

	MAP	RAP	RVS/RVD	PCWP	CO	SVR
Hypovolemic shock	50	2	20/2	4	2.2	1750
Cardiogenic shock	60	5	30/6	28	2.7	1620
Distributive shock (early sepsis)	55	4	30/3	5	8.6	473
Distributive shock (late sepsis)	60	6	28/6	7	2.8	540
Obstructive shock (PE)	75	9	40/8	10	3.8	1565
Obstructive shock (tamponade)	65	15	25/15	15	2.4	1648

MAP: mean arterial pressure; RAP: right atrial pressure; RVS: right ventricle systolic pressure; RVD: right ventricle diastolic pressure; PCWP: pulmonary capillary wedge pressure; CO/CI: cardiac output/ cardiac index; SVR: systemic vascular resistance.

Complications

- Hemorrhage: occult (hematoma formation) or overt bleeding from the site
- Vascular/vessel erosion
- Balloon rupture
- Atrial or ventricular dysrhythmias
- Nosocomial infections and septicemia
- Thromboembolic complications, thrombus in or around the catheter in the vessel.
- Venous air embolism
- Perforation of the cardiac chambers resulting in sudden circulatory collapse
- Pneumothorax
- Catheter knotting
- Ischemia/infarction of lung from prolonged balloon inflation while wedging, or mechanical occlusion of the pulmonary artery due to clot
- Rupture of a pulmonary artery segment. This should be suspected if either occult or life-threatening bleeding or air is aspirated from the distal PA port.

Troubleshooting the PAC

- If the hemodynamic numbers change suddenly or they do not seem to fit the clinical picture
  - Ensure that the measurements are being taken correctly.
  - Determine if the patient's position had recently been changed and adjust the transducer appropriately (should be level at mid-chest).
  - Ensure that zero-referencing is done accurately. Remove the protective caps from the transducer stopcock and position it such that the transducer stopcock should be off to the patient and should be open to air (i.e to atmospheric pressure)
  - Re-calibrate the monitors.
- If underwedged or overwedged waveform:
  - The *overwedged waveform* is characterized by a progressive rise in pressure during balloon inflation and results from the balloon trapping the tip against the vessel wall. If this occurs, the balloon should be deflated and the catheter repositioned.
  - The *underwedged waveform* occurs when the balloon is underinflated. Retract the balloon before re-inflating it
  - The *incompletely wedged waveform* is characterized by incomplete blood flow obstruction and is suspected when the PCWP is greater than the diastolic pulmonary artery pressure. The balloon needs to be retracted and re-inserted.

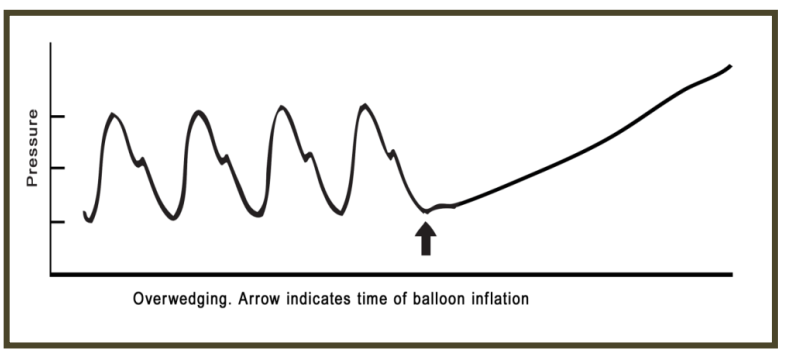


Figure 3. Overwedging.

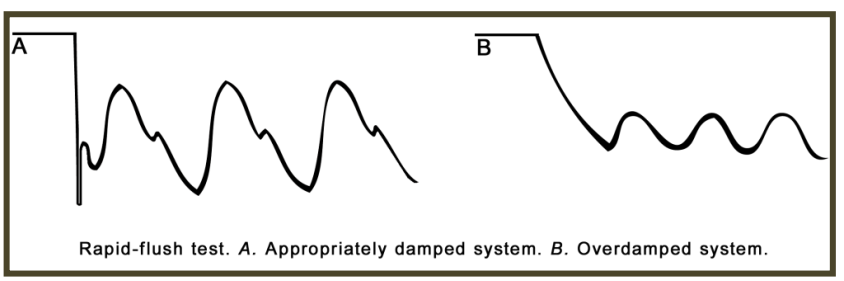


Figure 4. Troubleshooting a dampened waveform.

- Dampened PA waveform – (i.e lower PA systolic pressure and higher PA diastolic pressure)
  - Rule out shock or acute deterioration of the patient's clinical condition
  - Technical causes:
    - Problems in the PA catheter itself (PAC thrombus; knotted or kinked catheter; catheter migration or PAC abutting a vessel wall)
    - Problems with the external monitoring system (loose connections; air in the tubing; PAC thrombus; knotted or kinked tubing; inaccurate calibration)
- No PA waveform can be obtained
  - Problems with the PA catheter (ruptured or non-functional PAC balloon, or PAC thrombus)
  - Problems with the external monitoring system (loose connections; wrong stopcock position; amplifier is turned off; improper connection of the PA catheter transducer; non-functional transducer; defective cables; defective monitor; transducer dome that is broken, loose, or has air)
- Hematemesis or bloody secretion in ETT during PAC insertion:
  - This is an emergency as the patient is likely having pulmonary infarction/ rupture from prolonged balloon catheter inflation in the pulmonary artery. The catheter must be removed.

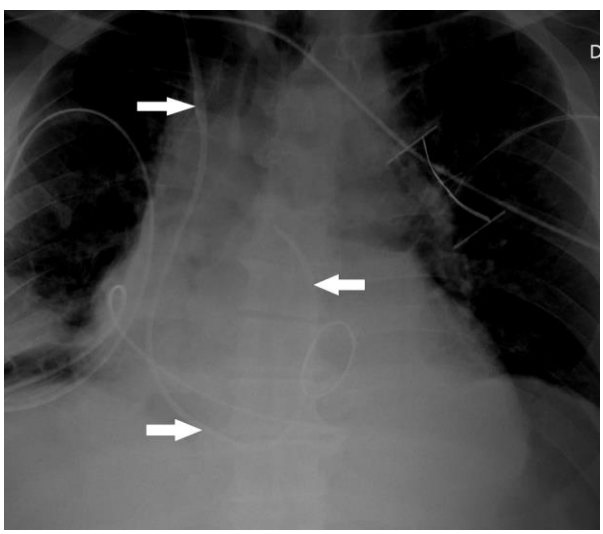


Figure 5. CXR confirmation of PAC placement.

References

G Edward Morgan Jr; Maged S. Mikhail; Michael J. Murray, *Clinical anesthesiology*, 3rd edition, 2002, Lange medical book/ Mcgraw Hill, p 166.

Marino PL. The Pulmonary Artery Catheter. In: Marino PL, ed. *The ICU Book*. 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2007: 163-181.

Rajaram SS, Desai NK, Kalra A, Gajera M, Cavanaugh SK, Brampton W, Young D, Harvey S, Rowan K. Pulmonary artery catheters for adult patients in intensive care. *Cochrane Database Syst Rev*. 2013 Feb 28;2:CD003408.

Vincent JL. Arterial, Central Venous, and Pulmonary Artery Catheters. In: Parrillo JE, Dellinger RP, eds. *Critical Care Medicine Principles of Diagnosis and Management in the Adult*. 3rd ed, Philadelphia, PA: Elsevier Health Sciences; 2008: 60-63.

Voyce SJ. Pulmonary Artery Catheters. In:Curley FJ, Heard SO, eds. *Irwin and Rippe's Intensive Care Medicine*. 7th ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2012: 57.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 22

Renal Replacement Therapy in the ICU

Rohan Arya, MD[1] , Rajaram Kandasamy, MD[2]* and Sri Sujanthy Rajaram, MD, MPH[3]
[1]Cooper Medical School of Rowan University, Camden, NJ, US
[2]Trinitas Regional Medical Center, Elizabeth, NJ, US
[3]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Acute renal failure is a common occurrence in critically ill patients, and greatly affects patient mortality and morbidity. In certain scenarios, renal replacement therapy (RRT) must be initiated.

When treating patients with acute kidney injury (AKI) in the ICU, there are several key questions that the clinician faces such as the optimal time of initiation of RRT, type of RRT and the frequency of this dialysis. Goals of RRT in critically ill patients are maintenance of electrolyte balance, solute removal and volume removal.

Critically ill septic patients often have significant intravascular volume depletion causing pre renal azotemia and volume repletion could improve the renal function.

Indications

Indications to initiate RRT can be divided into three parts: absolute indications; early vs. late initiation; and dialysis for intoxicants (see Table 1).

E-mail: rkandasmy@yahoo.com

Absolute Indications for RRT

- Intravascular volume overload that is not responsive to diuretics
- Refractory hyperkalemia or severe hyperkalemia with concerning EKG change
- Severe metabolic acidosis
- Uremia (encephalopathy, pericarditis, bleeding diathesis)

Early Start versus Delayed Initiation

Some experts recommend early initiation of RRT in cases where irreversible or persistent AKI is expected. There are several arguments against such an approach. First, it is unclear if early initiation would result in over-treating patients who would otherwise recover from AKI. The second uncertainty is how to define early versus late start: is urea level, urine output, or duration of AKI the standard to measure? The data supporting the benefit of early initiation of dialysis is mainly derived from studies with poor methodology and large heterogeneity. The overall effect is a significant decrease in 28-day mortality associated with early start (OR 0.45).

The decision to begin dialysis should take into account the likelihood of recovery from AKI, the duration of symptoms and, possibly, the overall prognosis of the patient.

Table 1. Example of intoxicants treated with hemodialysis

Intoxicant	Drug level	Other indications	Comment
Salicylates	> 80 mg/dL	CNS depression pulmonary edema or the condition of the patient warrants aggressive management.	Alkaline diuresis
Ethylene Glycol or Methanol	> 50 mg/dL	• Severe metabolic acidosis (pH < 7.25-7.30) • Renal failure • Visual symptoms/signs • Deteriorating vital signs despite intensive supportive care	Treat acidosis with sodium bicarbonate Initiate Fomepizole
Lithium	> 3.5 mmol/L > 2.5 mmol/L with symptoms		Level for dialysis differs for acute vs chronic intoxication
Valproic acid	N/A	Consideration when coma, severe liver dysfunction, or other organ failure.	Protein bound but when receptors saturated drug can be removed by RRT

Choice of Therapy

Several retrospective studies have suggested benefit from continuous therapy over intermittent hemodialysis (IHD). However, in a large randomized study there was no significant mortality difference between the two therapeutic options. Local expertise and

resource availability may dictate the modality selected. IHD can result in greater hemodynamic changes over a short period of time and may be a less desirable option in patients with liver failure or intracranial hypertension. Findings from recent studies indicate that there may be more functional recovery using continuous renal replacement therapy (CRRT).

Types of Continuous Therapy

Continuous arteriovenous hemofiltration (CAVH) uses an AV access to remove fluid and solutes via a semipermeable membrane. It relies on the patient's intrinsic blood pressure to drive fluid across the membrane. Since there is no diffusion, its hourly solute removal is low. This modality requires continuous, 24-hour therapy. CAVH is not commonly utilized in most Intensive Care Units (ICU).

Continuous venovenous hemofiltration (CVVH) is similar to CAVH except that a blood pump is required to drive fluid across the filter. The patient's blood pressure does not play a role in this therapy. Because it is pump-assisted, it achieves higher clearance of solutes and fluid. Continuous venovenous hemodialysis (CVVHD) can be combined with hemofiltration (CVVHDF). Patients' blood pressure does not play a role in therapy. These modalities can be used in hemodynamically unstable patients for solute and volume removal in Intensive care units and are commonly utilized in many ICUs.

Slow Continuous Ultrafiltration (SCUF) is used strictly to remove fluid. No substantial solute removal occurs. This can be used via AV or VV access.

Sustained Low Efficiency Daily Dialysis (SLEDD) is similar to IHD with slightly slower dialysate rates. Its major advantage to IHD is its hemodynamic stability. SLEDD is utilized in ICUs that does not have the capability of CVVHD.

Temporary dialysis catheters are placed at the femoral (20 cm catheter) or internal jugular veins (16 cm or smaller catheter). Subclavian vein should be avoided for temporary dialysis catheters.

Intensity of Treatment

There has been some debate as to whether more intense RRT would result in a survival benefit in critically ill patients. Two recent randomized trials clearly show that a dose of 20-30 mL/kg/hour of CRRT or IHD three times per week was sufficient and that more intense RRT would not result in benefits. One caveat is that with frequent interruption in RRT, it is recommended to aim for a higher goal of dialysis (such as 25% greater) in order to achieve the desired value.

Dialysis for Intoxication or Overdose

The decision to initiate RRT to remove a drug depends on several key factors: the size of the molecule incriminated in the volume of distribution, protein binding, hydrophilic characteristics, and clinical presentations. Digoxin is a prime example of medications not treated with RRT secondary to its high volume of distribution. Acetaminophen, on the other hand, can be dialyzed but NAC is considered treatment of choice.

References

Karvellas C. J., Farhat M. R., Sajjad I. et al. A comparison of early versus late initiation of renal replacement therapy in critically ill patients with acute kidney injury: a systematic review and meta-analysis. *Crit Care*. 2011; 15(1):R72.

Kellum J. A., Ronco C. Dialysis: Results of RENAL: What is the optimal CRRT target dose? *Nat Rev Nephrol*. 2010; 6(4):191-192.

Renal Replacement Therapy Study Investigators, Bellomo R., Cass A. et al. Intensity of continuous renal-replacement therapy in critically ill patients. *N. Engl. J. Med.* 2009; 361(17):1627-1638.

Ricci Z., Romagnoli S. Underdosing continuous venovenous hemofiltration predicts outcome in septic patients with acute kidney injury. *J Crit Care*. 2011; 26(2):221; author reply 222.

VA/NIH Acute Renal Failure Trial Network, Palevsky P. M., Zhang J. H. et al. Intensity of renal support in critically ill patients with acute kidney injury. *N. Engl. J. Med.* 2008; 359(1):7-20.

Vinsonneau C., Camus C., Combes A. et al. Continuous venovenous haemodiafiltration versus intermittent haemodialysis for acute renal failure in patients with multiple-organ dysfunction syndrome: a multicentrerandomised trial. *Lancet*. 2006; 368(9533):379-385.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 23

Targeted Temperature Management

Carol H. Choe, MD[1,*] and Sri Sujanthy Rajaram, MD, MPH[2]
[1]Cooper Medical School of Rowan University, Camden, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Therapeutic hypothermia, or targeted temperature management, is most often utilized after cardiac arrest, but has also been employed after neurologic insults including hypoxic-ischemic encephalopathy, hepatic encephalopathy, spinal cord injury, and ischemic CVA.

Research has demonstrated a benefit in hypoxic-ischemic pathologies, but further studies are being conducted to evaluate its use in spinal cord injuries, acute ischemic CVA, and traumatic brain injury. Targeted temperature management is also indicated in patients who have survived an out-of-hospital shockable cardiac arrest. Current guidelines recommend cooling to 32-34º Celsius. Cooling below 32ºC confers no mortality benefit, and may in fact increase adverse effects. Research is still ongoing to determine the optimal temperature to achieve maximal benefit. While data has been mixed for patients who suffered in-hospital and non-shockable cardiac arrests, most institutions recommend initiating hypothermic protocol for patients who do not have any neurologic recovery after their arrest.

Therapeutic hypothermia should be instituted as soon as possible after recognition of a cerebral ischemic event or cardiac arrest with poor neurologic recovery. During the induction phase, the patient is rapidly cooled (within 6-8 hours) to the targeted temperature. Once hypothermia has been achieved, the patient is maintained at this temperature for 24 hours. Rewarming commences slowly (0.5ºC per hour) until the patient is normothermic. Neurologic recovery after induced hypothermia may be delayed. Prognosis is deferred until at least 72 hours after the patient achieves normothermia and has been off sedation. Post rewarming

* Email: dr.carolchoe@gmail.com

hyperthermia should be avoided. Adequate sedation and paralytics can be used for mechanical ventilation and suppression of shivering during the induction phase.

Cooling may be achieved with ice packs to the head, axilla and groin; cold saline infusion; non-invasive cooling devices; or use of an endovascular cooling catheter. Due to their precision and ease of use, surface pads and intravascular catheters are most frequently utilized. They can rapidly cool the patient down to the targeted temperature, maintain temperature control, and rewarm the patient to normothermia. Cooling can be initiated and maintained while patient is undergoing diagnostic or therapeutic interventions such as cardiac catheterization.

Indications

- Poor neurologic recovery after return of spontaneous circulation
- Hypoxic brain injury, particularly perinatal hypoxic-ischemic encephalopathy

Potential Indications (few studies)

- Elevated intracranial pressure
- Spinal cord injuries
- Ischemic stroke
- Subarachnoid hemorrhage
- Hepatic encephalopathy

Contraindications

- Cardiac arrest due to drug overdose or trauma
- No return of spontaneous circulation in patients who suffered a cardiac arrest
- Glasgow Coma Scale (GCS) >8
- Pre-existing hypothermia
- Hemorrhagic stroke
- Coagulopathy and active bleeding
- Uncontrolled hemodynamically unstable dysrhythmias
- Severe hypotension not correctable by IV fluids, vasopressors, or invasive hemodynamic support (relative)
- Prolonged cardiac arrest >60 minutes (relative)
- Terminal condition or poor baseline status (relative)

Preparation

Patient set-up

- The patient should ideally be intubated and sedated.
- Shivering should be controlled if noted, Use of Meperidine, Magnesium, Opiates, paralytics

Equipment

- Cooling machine
- Surface cooling mats or endovascular cooling catheter
- Endovascular cooling catheter insertion kit (if utilized)
  - 500mL Normal Saline
  - Ultrasound
- Esophageal, bladder, or rectal temperature probe

Procedure

Cooling Mat application

1. Remove paper backing on the mats
2. Apply the mats to the torso and upper thighs, as indicated on the graphics
3. Attach the cords to the cooling machine
4. Set the desired temperature

Endovascular Catheter placement

Various catheters are available in the United States. The two most commonly used are the InnerCool catheter and the Alsius / Thermogard catheter. The Thermogard catheter is placed similarly to a central venous catheter. InnerCool catheter placement will be discussed here:

1. Place the appropriately-sized introducer catheter into the femoral vein (Please see Chapter 10)
2. The cooling device itself is a gold metallic catheter and should be sheathed within a "catheter hoop"
3. Attach a 20mL syringe filled with normal saline to the luer lock on the hoop; flush the saline through the hoop to lubricate the catheter and activate the heparin coating
4. Remove the catheter from the hoop
5. Remove the blue and yellow caps from the ports
6. Flush saline through one of the ports until it exits the other port
7. Hook these two luer ends together to create a loop
8. Remove the white cap and flush the port with saline; ensure it exits from the catheter

9. Using the xiphoid process as your marker, determine the length of catheter that will need to be placed in the patient
10. Gently guide the catheter through the cordis up to the predetermined marker
11. Aspirate blood from the middle port to ensure proper venous placement; flush the port with normal saline
12. Place the white cap back on the middle port
13. Obtain an X-Ray to confirm placement in the Inferior Vena Cava
14. Secure the catheter with a clear adhesive dressing
15. Connect the blue connector of the machine to the blue port on the catheter, and the yellow connector to the yellow port

Complications

- Complications related to line placement
- Cardiac dysrhythmias – Cooling lowers the heart rate
- Hypotension
- Hyperglycemia
- Cold-induced diuresis
- Electrolyte abnormalities – Low K, Mag, Phos during cooling
- Metabolic derangements
- Coagulopathy
- Seizures
- Pneumonia and sepsis
- Increased Lactate, Free fatty acids, Glycerol, Ketones and Osmolarity

Prognosis

Absence of corneal reflex, pupillary reaction and motor response by day 3, unresponsive to pain and discomfort by day 7, decerebrate rigidity by day 3 and status myoclonus are considered as poor prognostic markers.

References

Andrews PJD, Sinclair LH, Harris B, et al. Study of therapeutic hypothermia (32 to 35°C) for intracranial pressure reduction after traumatic brain injury (the Eurotherm3235Trial): outcome of the pilot phase of the trial. *Trials*. 2013;14:277.

Bosson N, Kaji AH, Niemann JT, et al. Survival and neurologic outcome after out-of-hospital cardiac arrest: results one year after regionalization of post-cardiac arrest care in a large metropolitan area. *Prehosp Emerg Care*. 2014;18(2):217–223.

Erb JL, Hravnak M, Rittenberger JC. Therapeutic hypothermia after cardiac arrest. *Am J Nurs*. 2012;112(7):38–44; quiz 46,45.

González-Ibarra FP, Varon J, López-Meza EG. Therapeutic hypothermia: critical review of the molecular mechanisms of action. *Front Neurol*. 2011;2:4.

Grulova I, Slovinska L, Nagyova M, Cizek M, Cizkova D. The effect of hypothermia on sensory-motor function and tissue sparing after spinal cord injury. *Spine J*. 2013;13(12):1881–1891.

Kim F, Nichol G, Maynard C, et al. Effect of prehospital induction of mild hypothermia on survival and neurological status among adults with cardiac arrest: a randomized clinical trial. *JAMA*. 2014;311(1):45–52.

Krieger DW, De Georgia MA, Abou-Chebl A, et al. Cooling for acute ischemic brain damage (cool aid): an open pilot study of induced hypothermia in acute ischemic stroke. *Stroke*. 2001;32(8):1847–1854.

Lyden PD, Hemmen TM, Grotta J, Rapp K, Raman R. Endovascular therapeutic hypothermia for acute ischemic stroke: ICTuS 2/3 protocol. *Int J Stroke*. 2014;9(1):117–125.

Marion D, Bullock MR. Current and future role of therapeutic hypothermia. *J. Neurotrauma*. 2009;26(3):455–467.

Nielsen N, Wetterslev J, Cronberg T, et al. Targeted temperature management at 33°C versus 36°C after cardiac arrest. *N. Engl. J. Med*. 2013;369(23):2197–2206.

Noyes AM, Lundbye JB. Managing the Complications of Mild Therapeutic Hypothermia in the Cardiac Arrest Patient. *J Intensive Care Med*. 2013.

Okada K, Ohde S, Otani N, et al. Prediction protocol for neurological outcome for survivors of out-of-hospital cardiac arrest treated with targeted temperature management. *Resuscitation*. 2012;83(6):734–739.

Oommen SS, Menon V. Hypothermia after cardiac arrest: beneficial, but slow to be adopted. *Cleve Clin J Med*. 2011;78(7):441–448.

Rivera-Lara L, Zhang J, Muehlschlegel S. Therapeutic hypothermia for acute neurological injuries. *Neurotherapeutics*. 2012;9(1):73–86.

Song SS, Lyden PD. Overview of therapeutic hypothermia. *Curr Treat Options Neurol*. 2012;14(6):541–548.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 24

Temporary Pacemakers

Adarsh Srivastava, MD[1], Carol Choe, MD[1], and Sri Sujanthy Rajaram, MD, MPH[2,*]
[1] Cooper Medical School of Rowan University, Camden, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Transvenous pacing involves placing of a catheter-based electrode into the right side of the heart. The procedure requires that central venous access be established through which the pacer catheter will be placed. Approach through the right internal jugular vein provides the most direct route to the right ventricle and is associated with the lowest complication rate. The pacemaker electrode is positioned in the right ventricle with the goal of pacing the endocardium.

Indications

Emergent Transvenous Pacemaker Placement

- Bradydysrhythmias
- Symptomatic sinus node dysfunction
  - Sinus Arrest
  - Sinus Bradycardia
- Symptomatic atrioventricular block
  - Second degree atrioventricular block, Mobitz type II
  - Third degree atrioventricular block

* Email: sujanty@gmail.com.

- Tachydysrhythmias
    - Overdrive pacing of rhythms refractory to medical management.
- Prophylactic Transvenous Pacemaker Placement
    - Acute myocardial infarction and
        - Symptomatic sinus node dysfunction
        - Second degree atrioventricular block, Mobitz type II
        - Third degree atrioventricular block
        - New left, right or alternating bundle branch block
        - New bi-fascicular block
    - Symptomatic patient secondary to failure of permanent pacemaker

Contraindications

Any contraindication to central venous catheter placement (See Chapter 10)

Preparation

Equipment needed:

- Introducer sheath
- Balloon tipped pacing catheter
    - 3 to 5 Fr Introducer
    - Approximately 100 cm in length with 10 cm interval markings
- External pacing generator which delivers the electrical current
    - Output control dial allows for the regulation of current that principally determines the ability of the pacemaker to "capture" the heart.
    - Rate control dial selects the pacing rate
    - Sensitivity control establishes a threshold, based on the amplitude of the native R wave, required to suppress the pacemaker from firing.
        - Turning the sensitivity control down will lead to a fixed rate or asynchronous pacing where the pacemaker fires regardless of the patient's underlying rhythm.
        - Increasing the sensitivity will lead to demand or synchronous pacing. The pacing generator will sense the intrinsic cardiac activity and inhibit the pacer from firing as long as the patient's heart rate is equal to or faster than the rate set on the pacing generator.
- Cardiac monitor

Procedure

After central venous access is obtained and introducer sheath is secured in place, the transvenous pacemaker can be inserted using cardiac monitoring.

1) Monitor the progression of the catheter as it approaches the right ventricle by analyzing the various waveforms that appear as the catheter is advanced. When utilizing a balloon tip pacing catheter, the balloon catheter should be inflated prior to progression into the right atrium.
2) A left bundle branch block pattern should be seen after every pacer spike.
3) Pace the chamber at 5-10 beats per minute above the patient's resting heart rate and slowly turn down the amplitude of the voltage/current delivered until capture is lost.
4) Set the final output 2-3 times the threshold to compensate for potential subsequent threshold elevations.
5) Sensitivity threshold testing can be checked only when there is spontaneous electrical activity.
   - Set the rate at 10-20 beats per minutes below the patient's resting heart rate and decrease the sensitivity (by increasing R wave cutoff) until pacing occurs.
   - Sensitivity threshold for the ventricle should be >6 mV
   - Set the final sensitivity 0.5 x R wave amplitude or 2 mV (whichever is higher)

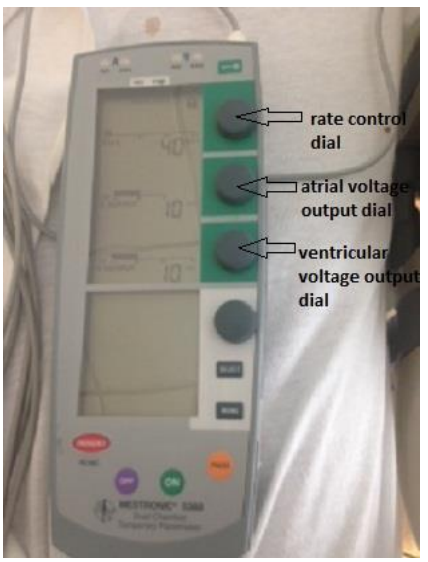
Complications

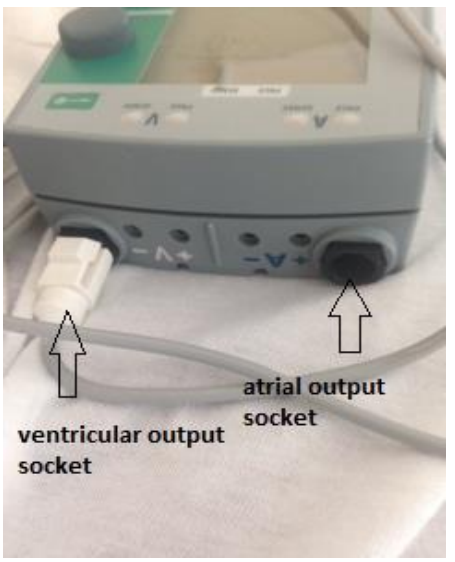
Complications of the Pacemaker

- Failure to pace – no pacing spike and no paced QRS complex seen
  - Causes
    - Over-sensing pacer is sensing noise as native QRS complexes and therefore does not pace. To solve this issue, reduce sensitivity
    - Battery or connection problem – Replace battery or re-attach connections
- Failure to Capture
  - Causes
    - Lead migration
    - Physiological alterations such as myocardial ischemia, hypoxia, acidosis, alkalosis, hyperglycemia, hypercapnea, drug administration.
    - Battery or connection problem
- Failure to sense – pacing spikes seen on top of native QRS complexes (R on T)
  - Causes
    - Lead migrationSensitivity set too low – increase sensitivity

Complications of Placement

- Venous Access
  - Pneumothorax
  - Hemothorax
  - Pseudoaneurysm
  - AV fistula
- Mechanical effects of lead/electrode
  - Promotion of ventricular ectopy
  - Right ventricular wall perforation → Can lead to tamponade
  - Complete heart block in patients with pre-existing left bundle branch block
- Infection
  - Highest risk when placed for >7 days





References

Bessman ES. Emergency cardiac pacing. In: Roberts JR, Hedges JR, eds. Clinical procedures in emergency medicine, 6th edn. Philadelphia, PA: *Saunders;* 2014:277–297

Harrigan RA, Chan TC, Moonblatt S, Vilke GM, Ufberg JW. Temporary transvenous pacemaker placement in the Emergency Department. *J Emerg Med.* 2007 Jan;32(1):105-11.

Jafri SM, Kruse JA. Temporary transvenous cardiac pacing. *Crit Care Clin* 1992;8:713–25

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 25

Thoracentesis

Rohan Arya[1*], MD, Sri Sujanthy Rajaram[2], MD, MPH and Archana Mishra[3], MD, MS*
Cooper Medical School of Rowan University, Camden, NJ, US[1]
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US[2]
State University of New York at Buffalo, Buffalo, NY, US[3]

Introduction

Thoracentesis is the recommended procedure of choice for diagnosing the type and possibly etiology of a pleural effusion. It can also be therapeutic if a large effusion is causing respiratory compromise. Pleural effusions can be caused by a variety of pathologies which are beyond the scope of this chapter. Determining the type of pleural effusion (which will likely shed light on the underlying pathology) is important in ruling out potential life threatening illnesses. Transudative effusion results from unbalanced hydrostatic and oncotic forces while exudative effusions result from inflammation, increased capillary permeability or lymphatic obstruction. In this chapter, the correct procedural approach to performing thoracentesis will be outlined.

Indications

- Pleural effusion of unknown cause
- Suspicion of infected pleural space
- Large Effusion causing respiratory distress

* Email: amishra@buffalo.edu

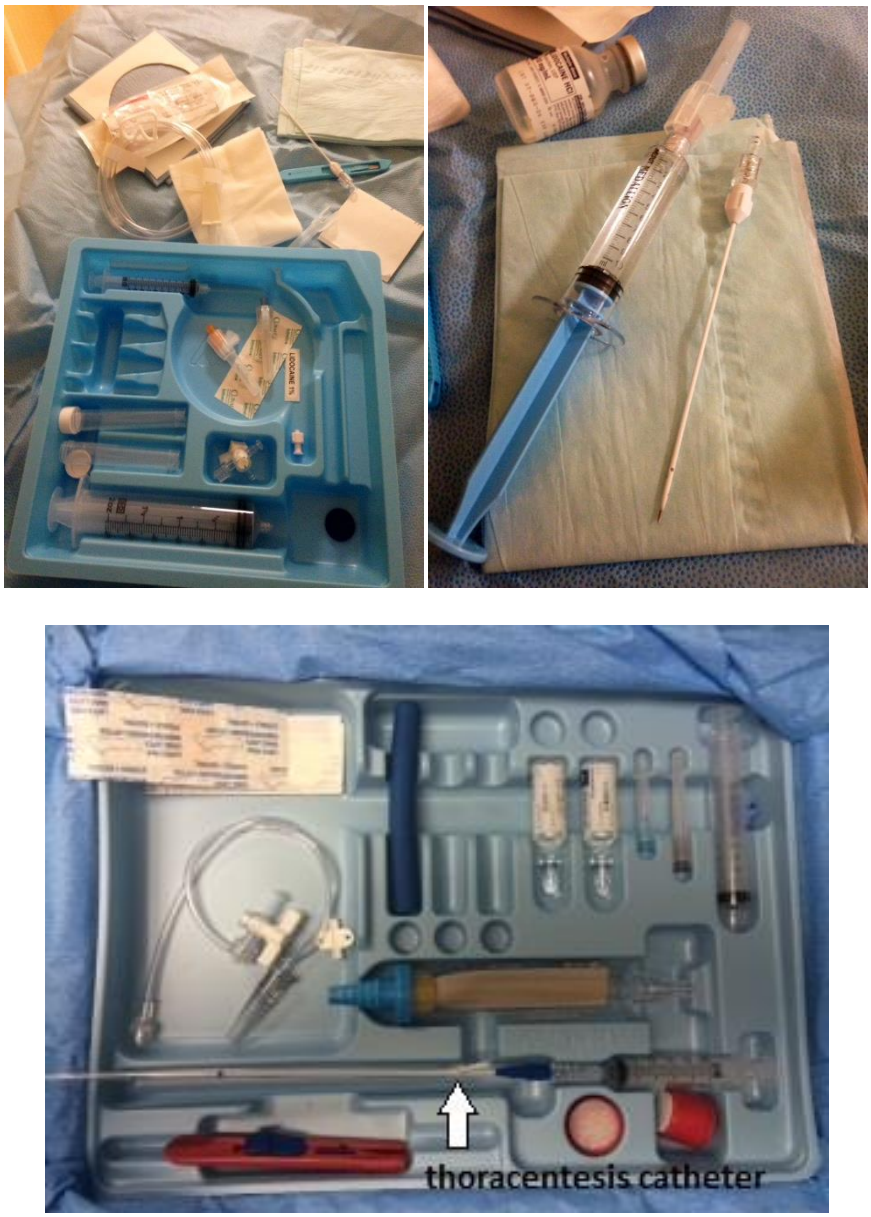


Figure. Standard thoracentesis kit.

Contraindications

- Clinical judgment should be used to ensure that the risk of performing the diagnostic procedure does not outweigh the benefits of obtaining the fluid sample.
- Coagulopathy: patient on anti-coagulation, thrombocytopenia or bleeding diasthesis.
- Needle should not pass through infected skin such as cellulitus or herpes zoster

- Blind insertion: ultrasound guidance during the procedure is considered the standard of care, especially in patients with risks of adhesions (previous empyema, previous thoracic surgery or previous pleurodesis.

Preparation

- Establish the indication for thoracentesis. Obtain consent from either the patient or surrogate. Evaluate for contraindications. Thoracentesis kits usually come in complete sets that contain the all the equipment and tools that will be used during the procedure.

Materials needed

- 18 gauge over-the-needle catheter
- a large syringe (35 to 60 ml)
- three way stopcock
- high pressure drainage tubing
- specimen tubes
- 1-2 large evacuated containers
- 1% lidocaine
- 25 and 22 gauge needle and small syringe for anesthetic injection
- Antiseptic solution such as chlorhexidine or povidone-iodine
- Sterile gloves, cap and gown
- Sterile drape
- Sterile occlusive dressing

Procedure

1. Thoracentesis is a sterile procedure so it is helpful to have one or two assistants to help position and monitor the patient. It is also helpful to have the additional help in collecting the fluid in the evacuated bottles and specimen tubes.
2. Verify the patient's identity, ensure that the site of procedure is correctly identified and take a time out immediately before the procedure for final verification by all members of the care team.
3. The patient must be positioned either sitting up on the edge of the bed, leaning forward with arms resting on a bedside table. If patient is unable to sit up then semi-reclined or supine position may be used.
4. The level of effusion can be estimated by dullness on percussion, absent fremitus and diminished or absent breath sounds on auscultation.
5. Using ultrasound, evaluate for a fluid collection. Mark a site from which the fluid can be accessed safely. The area should be prepped and draped in sterile fashion. The entire procedure should be performed under standard sterile precautions.

6. Over the proposed insertion site, using the 25-gauge needle, create a wheal on the epidermis with 1% lidocaine. Then insert a larger 22 gauge needle while maintaining negative pressure in the syringe, continue to attempt to anesthetize the proposed tissue tract overlying the superior edge of the rib that is below the selected intercostal space down to the pleura. Aspirate as you advance to rule out intravascular placement. Stop when pleural fluid is aspirated and inject additional lidocaine to anesthetize the sensitive parietal pleura. Note the depth of the needle and then remove it.
7. While maintaining negative pressure, insert the larger 18 guage introducer with the catheter over it into the same anesthetized tract. Avoid the inferior border of the rib to minimize injury to intercostal vessel and nerve. Once fluid is aspirated, feed the catheter into the pleural space.
8. Cover the hub of the catheter with your finger to prevent air from entering the pleural cavity.
9. Fasten the large syringe with the three way stopcock to the catheter hub after removing the introducer needle.
10. Aspirate about 50 ml of fluid for diagnostic purposes with the stopcock open to the patient and syringe. Make sure you close the stopcock to the patient after removing the required fluid.
11. If more fluid needs to be removed for therapeutic purpose one end of the high-pressure tubing should be attached to a port on the stopcock and the other end to a large evacuated container. Allow up to 1500 ml of fluid to drain with the stopcock open to the patient. Patient may start coughing or experience discomfort as more fluid is withdrawn and there is a risk of re-expansion pulmonary edema.
12. Aspirate adequate amount of fluid and place in appropriate specimen tubes:
    - Appearance: upon aspiration of the pleural fluid, its appearance will usually hint as to the cause of the effusion. Purulence and foul smelling fluid suggests an empyema. Grossly bloody fluid suggests hemothorax or malignancy. Milky effusions is concerning for chylothorax or empyema, and clear straw-colored fluid suggests a transudate.
    - Chemistry: LDH, glucose, pleural fluid total protein and pH should be ordered on all pleural fluid samples. Amylase, triglycerides and cholesterol maybe ordered if there is a suspicion for GI causes and chylothorax, respectively.
    - Send simultaneous serum LDH and protein to allow distinction between transudate and exudate.
    - Cell Counts and Cytology: cell count with differential will aid in determining if the pleural effusion is potentially infected and may help to determine other possible causes. Cytology is done to evaluate for malignancy.
    - Microbiological Stains and Cultures: this should always be performed if the patient is febrile, septic or if there is any suspicion of an infectious process.
13. Remove the thoracentesis catheter at end expiration and place an occlusive sterile dressing over the insertion site. Remove any antiseptic lotion from the skin.
14. Place all sharp and contaminated objects in appropriate safety containers.

Complications

- Pneumothorax
- Bleeding
- local pain

Interpretation

The Light's Criteria: Differentiates transudative and exudative pleural effusions. If one of the below is present, the pleural fluid is an exudate.

Pleura fluid (PF) protein/Serum protein ratio > 0.5 PF LDH/serum LDH > 0.6 PF LDH > two-thirds the upper limit of normal laboratory value.

The Modified Light's Criteria

Pleural fluid protein > 2.9 g/dL Pleural fluid cholesterol > cholesterol 45 mg/dL Pleural fluid LDH > 0.45 times the upper limit of normal laboratory value

References

Grogan DR, Irwin RS, Channick R, Raptopoulos V, Curley FJ, Bartter T, et al. Complications associated with thoracentesis: a prospective, randomized study comparing three different methods. *Archives of internal medicine.* 1990;150(4):873-7.

http://www.uptodate.com/contents/diagnostic-thoracentesis.

Laws D, Neville E, Duffy J. BTS guidelines for the insertion of a chest drain. *Thorax.* 2003;58(suppl 2):ii53-ii9.

Light RW. Pleural effusions.Medical *Clinics of North America*. 2011;95(6):1055-70.

Sahn SA. The differential diagnosis of pleural effusions.*Western Journal of Medicine.* 1982;137(2):99.

Sokolowski Jr JW, Burgher LW, Jones Jr FL, Patterson JR, Selecky PA.Guidelines for thoracentesis and needle biopsy of the pleura.*American Review of Respiratory Disease.* 1989;140(1):257-8.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 26

Transcranial Doppler Monitoring

M. Kamran Athar, MD
Thomas Jefferson University, Philadelphia, PA, US

Transcranial Doppler (TCD) ultrasound uses low frequency (2 MHz) pulsed sound to penetrate bony windows and visualize intracranial vessels of the circle of Willis. Its use has gained wide acceptance in stroke and neurologic intensive care units as a noninvasive means of assessing the patency of intracranial vessels.

Indications for TCD

Detection of Cerebral Vasospasm in SAH

TCD is useful for detecting and monitoring vasospasm in Subarachnoid Hemorrhage (SAH). Changes in mean flow velocity detected by TCD can precede the clinical sequelae of vasospasm. Daily recording of TCD velocities offer a window of opportunity to treat patients prior to clinical decline.

Lindegaard et al. reported a Middle Cerebral Artery (MCA) blood flow velocity of more than 120 cm/sec is suggestive of vasospasm. They also showed that an MCA velocity above 200 cm/sec in patients with vasospasm is indicative of a residual lumen diameter reduction of 1 mm. The Lindegaard ratio — mean MCA velocity / mean ICA velocity — of greater than 3.0 indicates the presence of vasospasm, and the ratio of >6 indicates severe MCA vasospasm [1].

Diagnosis of Brain Death

TCD showing small systolic peaks without diastolic flow or a reverberating flow pattern suggests high vascular resistance and supports the diagnosis of brain death (Figure 1).

Limitations include a 10-25 percent prevalence of temporal bone thickening that precludes evaluation of 6 of the usual 11 insonated intracranial arteries. Because of these and other technical limitations, absence of arterial signals on TCD (a finding in 9 percent of brain dead patients) is considered nondiagnostic.

In one study comparing 61 patients with clinical brain death with 39 controls, the sensitivity of TCD was 70.5 percent, and the specificity was 97.4 percent [2].

Similar results were observed in a case-control study of 101 comatose patients in which it was also observed that both sensitivity and specificity improved over time to 100 percent for examinations performed 24 hours or more after clinical diagnosis of brain death [3].

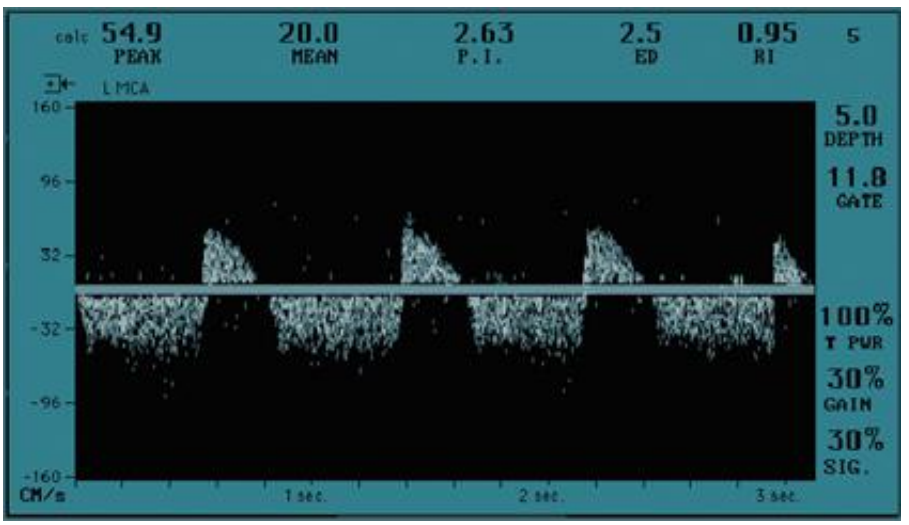


Figure 1.

Acute Ischemic Stroke

In patients with acute stroke, TCD is able to detect intracranial stenosis, identify collateral pathways, detect emboli on a real-time basis, and monitor reperfusion after thrombolysis. [4,5,6]

Sickle Cell Disease (SCD)

TCD has become an important tool in predicting risk for stroke in patients with SCD. It measures the time-averaged mean velocity of blood flow in the large intracranial vessels, which is inversely related to arterial diameter (as documented by cerebral angiography). [7]

A focal increase in velocity usually suggests arterial stenosis, whereas a bilateral increase may indicate bilateral arterial disease, increased blood flow, or both. [8,9]

In children, a mean transcranial Doppler (TCD) velocity >170 cm/sec is worrisome, and values >200 cm/sec in the middle cerebral or internal carotid artery are highly associated with

an increased risk of stroke, even before lesions become evident on magnetic resonance angiograms (MRA). [10]

Patients with abnormal MRA findings and higher TCD velocities are at even higher risk for stroke.

This association was illustrated in a study in which TCD was performed in 190 children with SCD (age at entry: 3 to 18 years). [11]

Twenty-three patients (12 percent) had an abnormal TCD (based upon the highest blood flow velocity in the middle cerebral artery), and seven developed a cerebral infarction after an average follow-up of 29 months. Six of the seven strokes occurred among the 23 patients with abnormal ultrasound results [12].

Limitations

Major drawbacks include operator-dependence, poor patient windows (unable to insonate a flow signal in 15 percent of cases), and low sensitivity in the vertebrobasilar system.

References

[1] Lindegaard, KF; Nornes, H; Bakke, SJ; Sorteberg, W; Nakstad, P. Cerebral vasospasm diagnosis by means of angiography and blood velocity measurements. *Acta Neurochir* (Wien), 100, 12–24, 1989

[2] Dosemeci, L; Dora, B; Yilmaz, M; et al. Utility of transcranial doppler ultrasonography for confirmatory diagnosis of brain death: two sides of the coin. *Transplantation*, 2004, 77, 71.

[3] Kuo, JR; Chen, CF; Chio, CC; et al. Time dependent validity in the diagnosis of brain death using transcranial Doppler sonography. *J Neurol Neurosurg Psychiatry*, 2006, 77, 646.

[4] Alexandrov, AV; Demchuk, AM; Wein, TH; Grotta, JC. Yield of transcranial Doppler in acute cerebral ischemia. *Stroke*, 1999, 30, 1604.

[5] Gao, S; Wong, KS; Hansberg, T; et al. Microembolic signal predicts recurrent cerebral ischemic events in acute stroke patients with middle cerebral artery stenosis. *Stroke*, 2004, 35, 2832.

[6] Saqqur, M; Shuaib, A; Alexandrov, AV; et al. Derivation of transcranial Doppler criteria for rescue intra-arterial thrombolysis: multicenter experience from the Interventional Management of Stroke study. *Stroke*, 2005, 36, 865.

[7] Adams, RJ; Nichols, FT; Figueroa, R; et al. Transcranial Doppler correlation with cerebral angiography in sickle cell disease. *Stroke*, 1992, 23, 1073.

[8] Adams, RJ; McKie, VC; McKie, K; et al. Improving transcranial Doppler (TCD) predictive criteria for ischemic stroke. *Proceedings of the 18th Annual Meeting of the Sickle Cell Program*, Philadelphia, May 1993.

[9] Verlhac, S; Bernaudin, F; Tortrat, D; et al. Detection of cerebrovascular disease in patients with sickle cell disease using transcranial Doppler sonography: correlation with MRI, MRA and conventional angiography. *Pediatr Radiol*, 1995, 25 Suppl 1:S14.

[10] Abboud, MR; Cure, J; Granger, S; et al. Magnetic resonance angiography in children with sickle cell disease and abnormal transcranial Doppler ultrasonography findings enrolled in the STOP study. *Blood*, 2004, 103, 2822.

[11] Adams, R; McKie, V; Nichols, F; et al. The use of transcranial ultrasonography to predict stroke in sickle cell disease. *N Engl J Med*, 1992, 326, 605.

[12] Alexandrov, AV; Demchuk, AM; Wein, TH; Grotta, JC. Yield of transcranial Doppler in acute cerebral ischemia. *Stroke*, 1999, 30, 1604.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 27

Ultrasound in the Intensive Care Unit (ICU)

David Shrift, MD[2] and Sri Sujanthy Rajaram, MD, MPH[1,*]
[1]Critical Care Medicine, JFK Medical Center, Edison, NJ, US
[2]University of South Carolina School of Medicine, Columbia, SC, US

Introduction

This ultrasound manual is designed to show you how ultrasound can improve patient care in the ICU. The applications highlighted will include evidence to support their use and key aspects to the scan. This manual is NOT designed to teach you how to use the ultrasound.

Evaluation of Inferior Vena Cava (IVC) Compressibility

Uses:

- Determining volume responsiveness
- Helps in the assessment of the hypotensive patient

Evidence:

- IVC collapsibility > 12-18% measured just distal to the hepatic vein predicts cardiac index (CI) will increase > 15% in response to 7-8mL/kg of colloid with > 90% PPV and NPV. This only applies to mechanically ventilated patients without spontaneous breaths on VC at 8-10mL/kg with minimal PEEP and patients without arrhythmias.

* Email: sujanty@gmail.com.

- Goal-directed ultrasound for the hypotensive patient involves an IVC assessment to help distinguish among the multiple causes of shock.

Compression Ultrasound to Diagnose Deep Vein Thrombosis (DVT)

Uses:

- Determine the presence of a DVT
- Helps to avoid placing central lines into veins with DVTs
- Assists in the decision to give thrombolytic to the unstable patient with suspected PE

Evidence:

- A recent meta-analysis demonstrates emergency medicine (EM) physicians have excellent accuracy (> 95% sensitivity and specificity) to detect the presence of a DVT using portable ultrasound.

Key Aspects:

- Requires some practice with the ultrasound
- Compression of the saphenous, common femoral and popliteal vein is sufficient
- Only evaluates for proximal DVTs
- Most useful when traditional ultrasound is not possible
- Go to http://www.ultrasoundpodcast.com/2011/08/dvt/ for an excellent podcast on this topic

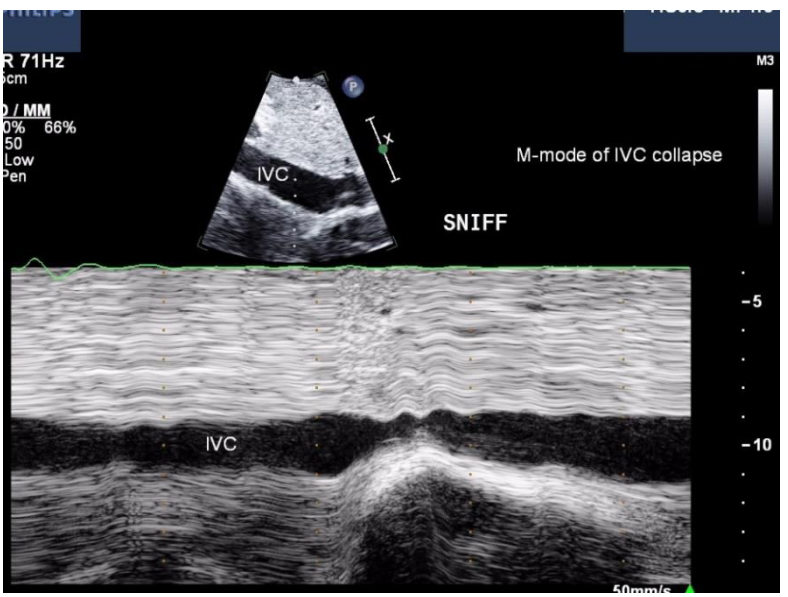


Figure 1. Evaluating IVC Compressibiity.

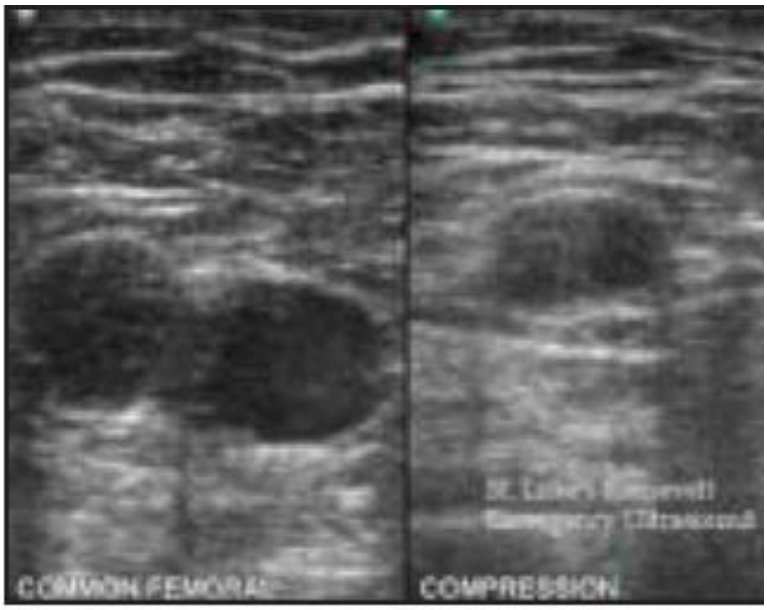


Figure 2. Evaluation of DVT.

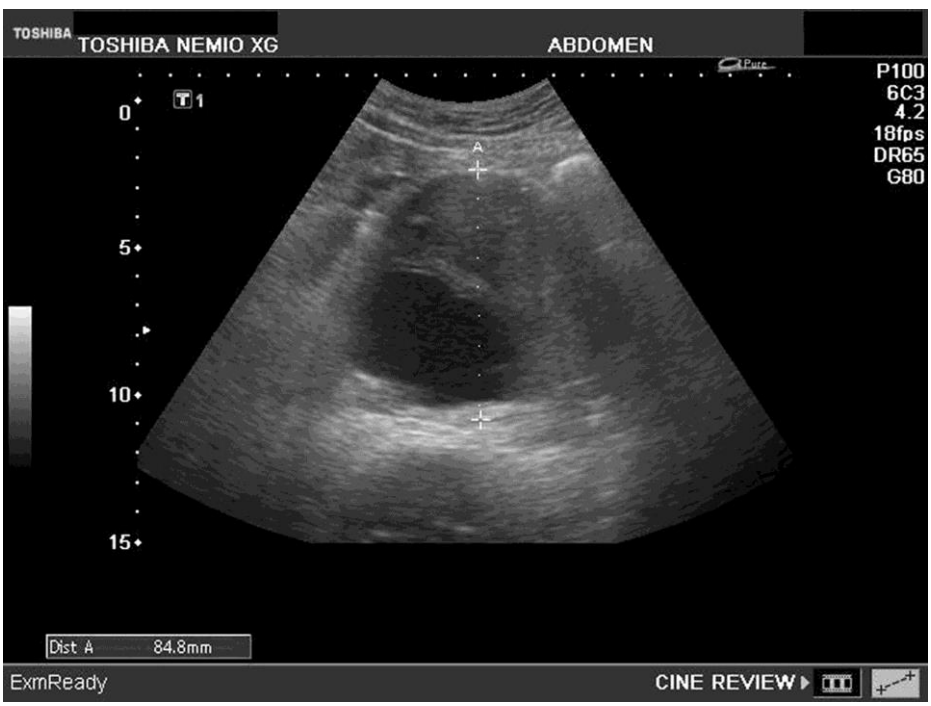


Figure 3. Evaluation of the aorta.

Evaluation of the Aorta

Uses:

- Assess for abdominal aortic aneurysm (AAA)
- Part of the focused goal directed ultrasound exam for undifferentiated hypotension.

Evidence:

- EM physician based aortic ultrasound has near perfect accuracy for the detection and/or exclusion of AAA.

Key Aspects:

- This can is most useful in the undifferentiated shock patient
- Perform scan in the short-axis
- Scan from celiac trunk to aortic bifurcation
- Bowel gas can be overcome by steady pressure

References

Barbier C., Loubières Y., Schmit C. et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med*. 2004; 30(9):1740-1746.

Burnside P. R., Brown M. D., Kline J. A. Systematic review of emergency physician-performed ultrasonography for lower-extremity deep vein thrombosis. *Acad Emerg Med*. 2008; 15(6):493-498.

Dent B., Kendall R. J., Boyle A. A., Atkinson P. R. T. Emergency ultrasound of the abdominal aorta by UK emergency physicians: a prospective cohort study. *Emerg Med J*. 2007; 24(8):547-549.

Feissel M., Michard F., Faller J.-P., Teboul J.-L. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med*. 2004; 30(9):1834-1837.

Knaut A. L., Kendall J. L., Patten R., Ray C. Ultrasonographic measurement of aortic diameter by emergency physicians approximates results obtained by computed tomography. *J Emerg Med*. 2005; 28(2):119-126.

Perera P., Mailhot T., Riley D., Mandavia D. The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically lll. *Emerg. Med. Clin. North Am.* 2010; 28(1):29-56, vii.

Tayal V. S., Graf C. D., Gibbs M. A. Prospective study of accuracy and outcome of emergency ultrasound for abdominal aortic aneurysm over two years. *Acad Emerg Med*. 2003; 10(8):867-871.

In: Critical Care Procedure Book
Editors: Sri Sujanthy Rajaram
ISBN: 978-1-63482-405-7
© 2015 Nova Science Publishers, Inc.

Chapter 28

Venous Oxygen Saturation Monitoring

Philip Willsie, MD[1], and Sri Sujanthy Rajaram, MD, MPH[2*]
[1]Cooper Medical School of Rowan University, Camden, NJ, US
[2]Critical Care Medicine, JFK Medical Center, Edison, NJ, US

Introduction

Venous oxygen saturation monitoring is used to evaluate the adequacy of oxygen supply and demand, and to prognosticate and titrate therapies in shock states. Venous oxygen saturation is often an unreliable indicator of the presence or absence of tissue hypoxia, particularly in the setting of sepsis. Studies have shown that it often does not correlate with simultaneously measured lactate levels. There are many confounding factors that influence venous oxygen saturation levels. Thus, controversy exists regarding the usefulness of measuring venous oxygen saturation in septic patients. Two types of monitors are frequently employed: mixed venous and central venous oxygen saturation.

$ScvO_2$ (central venous oxygen saturation) measures the oxygen saturation of hemoglobin at the superior vena caval-right atrial junction. It is calculated from a sample of blood taken from a central venous catheter or continuously measured and displayed on a bedside monitor. A normal range is 75-80% ± 5-6% for spontaneous variation, and may be 5-8% higher than SvO_2 in septic shock patients. It can vary from simultaneously measured SvO_2 by as much as 10%. Thus, the average of multiple values is often used.

SvO_2 (mixed venous oxygen saturation) measures the oxygen saturation of hemoglobin in the pulmonary artery. It is calculated from a sample of blood taken from the distal port of the pulmonary artery catheter or continuously measured and displayed on a monitor at the bedside. A normal range is 70-75% ± 5-6%. It provides more information than $ScvO_2$ by incorporating myocardial oxygenation. As myocardial oxygen consumption increases, the

* Email: sujanty@gmail.com.

difference between $ScvO_2$ and SvO_2 may increase. Measurement requires placement of a pulmonary artery catheter, which may be more invasive and prone to certain risks.

$SvO_2 = DO_2/VO_2$

DO_2 = Oxygen Delivery (mL of O_2/min) = (Hgb x 1.34 x CO x 10)

Normal value = 520-600 mL/min/m^2

Options to improve O_2 delivery are to increase:

Hgb, cardiac output (CO), or SaO_2

VO_2 = Oxygen Uptake (mL of O_2/min) =

(Hgb x 13.8 x CO x 10) x ($SaO_2 – SvO_2$)

Normal value = 250mL/min. VO_2 <100 mL/min suggests impaired tissue oxygenation. Essentially same as O_2 consumption by tissue since O_2 not stored in tissue

Indications

Surrogate for Cardiac Output

Assuming hemoglobin (Hgb), arterial oxygen saturation (SaO_2) and oxygen consumption are stable, the contribution of the partial pressure of oxygen (PaO_2) is negligible. The SvO_2 may change in proportion to the change in cardiac output (CO).

Decreased CO → decreased O_2 delivery → increased O_2 extraction → decreased SvO_2

Shock

A clinical state resulting from of global tissue dysoxia (when metabolic production is limited by the supply or utilization of oxygen). It can be secondary to inadequate delivery, extraction or utilization of oxygen.

Cardiac

SvO_2 has diagnostic, prognostic and therapeutic value in acute myocardial infarction (AMI). After AMI, SvO_2 <60% was strongly associated with CHF and mortality, and decrease in SvO_2 >5% always showed significant decrease in cardiac index (CI). SvO_2 was found to be strongly correlated with CI.

Sepsis

According to the Surviving Sepsis Campaign, patients should have a goal SvO_2 >65% (or $ScvO_2$ >70%) in first 6 hours. $ScvO_2$ has been shown to improve mortality when used as part

of an overall therapy (lactate measurement, blood cultures before antibiotics, broad spectrum antibiotics, fluid, vasopressors, CVP >8, and $ScvO_2$ >70%). SvO_2 <70% correlates strongly with mortality after 48 hours in septic shock. $ScvO_2$ ≥70% + lactate clearance of ≥20% in 2 hours for first 8 hours showed a 9.6% decreased mortality in severe sepsis. Although SvO_2 may initially be low in septic shock, after initial resuscitation it is often normal or elevated despite organ dysfunction. Thus, a high $ScvO_2$ in the setting of septic shock can be falsely reassuring.

Surgery/Trauma

In patients with suspected blood loss, $ScvO_2$ was superior to normal parameters at predicting blood loss. After brain injury, $ScvO_2$ < 65% in the first 24 hours had a higher mortality and longer length of stay. Intraoperative $ScvO_2$ <70% was associated with postoperative complications. Post-operatively, a low $ScvO_2$ in first 8 hours was associated with post-operative complications and a higher 28-day mortality.

Contraindications

- Contraindications to CVC or PA catheter placement

Interpretation

- SvO_2 > 75% represents normal extraction
- SvO_2 50-75% represents compensated extraction with increased O_2 demand or decreased O_2 supply
- SvO_2 30-50% represents limited extraction with a greater discrepancy between O_2 supply and demand
- 50% is the theoretical lower limit of SvO_2 and $ScvO_2$, which indicates global tissue dysoxia
- Actual (significant or real) change must be >5% for >10 minutes
- Continuous measurement is accurate to within 1.5% of laboratory measurement

Possible Causes of Low Value

- Inadequate CO
- Inadequate hemoglobin
- Left shifted O_2-Hgb dissociation curve: hypothermia, alkalosis, low 2,3-DPG concentration, methemoglobinemia, carbon monoxide poisoning
- Inadequate SaO_2
- Increased tissue aerobic metabolism
- Respiratory burst of systemic inflammation consuming oxygen

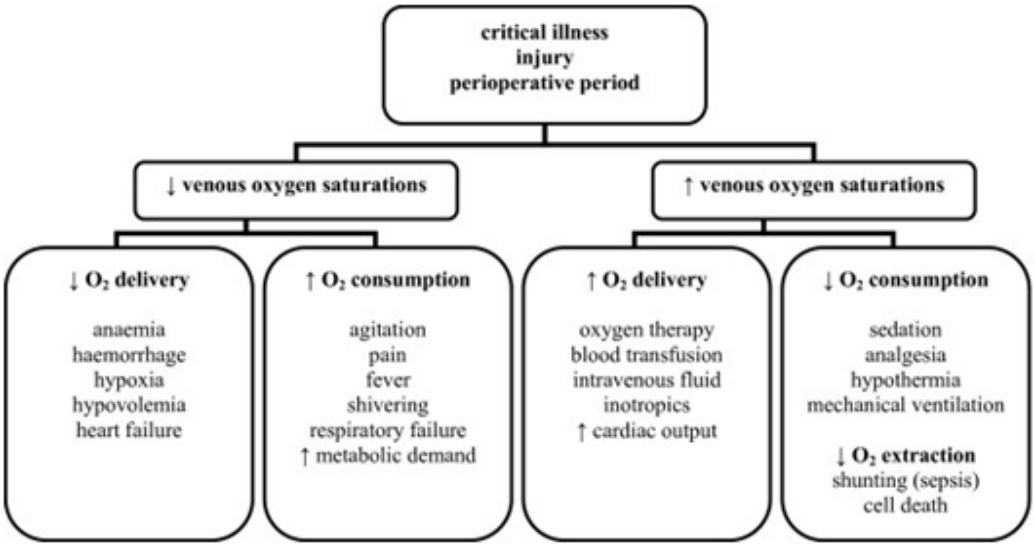


Figure 1. Differential diagnoses for changes in venous oxygen saturation.

References

Ander DS, Jaggi M, Rivers E, et al. Undetected cardiogenic shock in patients with congestive heart failure presenting to the emergency department. *Am J Cardiol* 1998;82:888-91.

Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2012. *Crit Care Med.* 2013;41(2):580-637.

Di Filippo A, Gonnelli C, Perretta L, et al. Low central venous saturation predicts poor outcome in patients with brain injnury after major trauma: a prospective observational study. *Scand J Trauma Resusc Emerg Med* 2009;17:17-23.

Edwards JD. Oxygen transport in cardiogenic and septic shock. *Crit Care Med.* 1991 19;(5):658-63.

Futier E, Robin E, Jabaudon M, et al. Central venous O_2 saturation and venous-to-arterial CO_2 difference as complementary tools for goal-directed therapy during high-risk surgery. *Crit Care* 2010;14(5):R193.

Ho KM, Harding R, Chamberalin J, Bulsara M. A comparison of central and mixed venous oxygen saturation in circulatory failure. *J Cardiothoracic and Vasc Anesthesia* 2010;24(3):434-439.

Jansen TC, van Bommel J, Schoonderbeek FJ, et al. LACTATE study group: Early lactate-guided therapy in intensive care unit patients: A multicenter, open-label, randomized controlled trial. *Am J Respir Crit Care Med* 2010;182:752–761.

Kan K, Koeda T, Ichikawa T, et al. Relation between mixed venous blood oxygen saturation and cardiac pumping function at the acute phase of myocardial infarction. *Jpn Circ J.*1989;53(12):1481-90.

Kopterides P, Bonovas S, Marvou I, et al. Venous oxygen saturatio and lactate gradient from superior vena cava to pulmonary artery in patients with septic shock. *Shock* 2009;36(6):561-67.

Marino PL. *The ICU Book*. Baltimore, MA: Williams & Wilkins; 2007.

Nebout S, Pirracchio R. Review Article: Should We Monitor ScVO2 in Critically Ill Patients? *Cardiol Research and Practice* 2012,Article ID 370697, 7pages.

Park J, Lee J, Park YS, et al. Prognostic Value of Central Venous Saturation and Blood Lactate Levels Measured Simultaneously in the Same Patients with Severe Inflammatory Response Syndrome and Severe Sepsis. *Crit Care* 2014;192:435-440.

Pearse R, Dawson D, Fawcett J, et al. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomized controlled trial. *Crit Care* 2005;9:R687-93.

Polonen P, Ruokonen E, Hippelainen M, et al. A prospective, randomized study of goal-orientated hemodynamic therapy in cardiac surgery patients. *Anesth Analg* 2000;90:1052-1059.

Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368-77.

Rivers EP, Martin GB, Smithline H et al. The clinical implications of continuous central venous oxygen saturation during human CPR. *Ann Emerg Med* 1992;21:1094-1101.

Scalea TM, Harnett RW, Duncan AO, et al. Central venous oxygen saturation: a useful clinical tool in trauma patients. *J Trauma*. 1990;30:1539-43.

Varpula M, Tallgren M, Saukkonen K, Voipio-Pulkki LM, Pettilä V. Hemodynamic variables related to outcome in septic shock. *Intensive Care Med*. 2005;31(8):1066-71.

Van Beest P, Wietasch G, Scheeren T, et al. Clinical review: use of venous oxygen saturations as a goal – a yet unfinished puzzle. *Crit Care* 2011;15:232.

Index

A

acid, 13, 140
acidosis, 70, 90, 140, 151
acute asthma, 114
acute renal failure, 142
adhesions, 116, 155
adolescents, 19
adult respiratory distress syndrome (ARDS), 43, 91
adults, 69, 81, 92, 100, 147
adverse effects, 143
air embolism, 17, 135
airways, 11, 13, 89, 90, 112
albumin, 118, 119, 120
alkalosis, 151, 169
alveoli, 111
amplitude, 85, 86, 150, 151
anatomy, 4, 24, 122, 123, 128
anchoring, 5
anesthesiologist, 92
anesthetization, 14
aneurysm, 74, 166
angina, 78
angiography, 160, 161, 162
angulation, 65
antibiotic, 46
anticoagulant, 43
anticoagulation, 42, 124
antihypertensive agents, 2
aorta, 40, 41, 56, 73, 75, 117, 165, 166
aortic regurgitation, 77
aortic ultrasound, 166
aortic valve, 73, 75, 76, 77, 85
apex, 4, 57
apnea, 93, 94
arrest, 63, 94, 143, 144, 146
arrhythmia, 74, 75, 94
arterial blood gas, 1, 41
arteries, 1, 2, 3, 17, 74, 99, 130, 160
artery, 2, 3, 4, 5, 6, 15, 17, 19, 41, 74, 75, 101, 117, 122, 128, 131, 132, 133, 134, 135, 137, 138, 160, 161, 167, 171
ascites, 69, 115, 117, 118, 120
aseptic, 17, 70, 132
aspirate, 18, 65
aspiration, 13, 48, 67, 90, 91, 93, 113, 116, 127, 156
assessment, 43, 55, 57, 58, 61, 71, 90, 123, 128, 132, 163, 164
asthma, 90
asymptomatic, 52
atelectasis, 11
atmospheric pressure, 136
atria, 57
atrioventricular block, 149, 150
atrium, 59
auscultation, 24, 155
autonomy, ix
axilla, 4, 144
azotemia, 139

B

bacillus, 13
base, 90, 101
benefits, 141, 154
betadine, 106
bicarbonate, 140
Bilateral, 116
biopsy, 32, 157
bleeding, 5, 12, 18, 20, 25, 28, 33, 34, 35, 37, 39, 47, 116, 117, 122, 123, 127, 130, 135, 140, 144, 154
blood, 1, 2, 3, 4, 5, 13, 15, 17, 18, 19, 20, 39, 40, 41, 42, 43, 69, 71, 73, 75, 78, 81, 99, 102, 111, 113,

114, 116, 117, 123, 124, 133, 136, 141, 146, 159, 160, 161, 167, 169, 170
blood cultures, 169
blood flow, 3, 5, 20, 41, 71, 73, 99, 133, 136, 159, 160, 161
blood pressure, 1, 2, 4, 75, 76, 111, 124, 141
blood supply, 2
blood vessels, 123
bloodstream, 21
bone, 24, 63, 64, 65, 89, 107, 160
bone marrow, 64, 65
bowel, 117, 118
bowel perforation, 117
bradycardia, 13, 81, 95
brain, 7, 8, 9, 81, 85, 86, 99, 100, 102, 144, 147, 159, 160, 161, 169, 170
brain activity, 99
brain damage, 147
brain herniation, 81, 86
brainstem, 102
branching, ix
breathing, 20, 60, 90, 91, 111, 112
bronchial tree, 11
bronchoscopy, 11, 14, 92, 97, 121, 123, 128, 130
bronchospasm, 12
bundle branch block, 150, 151, 152

C

cables, 8, 11, 132, 137
cadaver, 130
caliber, 20
calibration, 137
capillary, 153
carbon, 169
carbon monoxide, 169
cardiac activity, 150
cardiac arrest, 20, 42, 95, 143, 144, 146, 147
cardiac catheterization, 144
cardiac output, 41, 76, 79, 111, 132, 134, 135, 168
cardiac structure, 52, 53, 55
cardiac surgery, 171
cardiac tamponade, 61
cardiogenic shock, 42, 73, 74, 80, 170
cardiopulmonary bypass, 100
caregivers, 84
cartilage, 18, 24, 96
catheter, 2, 3, 4, 5, 7, 8, 15, 16, 17, 18, 19, 20, 21, 27, 30, 31, 40, 70, 71, 72, 74, 75, 84, 87, 92, 100, 101, 102, 116, 117, 131, 132, 133, 134, 135, 136, 137, 141, 144, 145, 146, 149, 150, 151, 155, 156, 167, 169
catheterizations, 131
cellulitis, 64, 116
central nervous system (CNS), 109
cephalosporin, 46
cerebellum, 102
cerebral blood flow, 81, 82, 93, 99
cerebral edema, 83, 108
cerebral hypoxia, 102
cerebrospinal fluid, 84, 105
cerebrovascular disease, 161
certification, ix
children, ix, 19, 24, 63, 160, 161, 162
cholesterol, 156, 157
chronic obstructive pulmonary disease (COPD), 90, 114
chylothorax, 156
chylous ascites, 118
circulation, 2, 3, 39, 144
cirrhosis, 28, 117, 120
classification, 90
claustrophobia, 113
clavicle, 19, 63, 92
cleaning, 11, 17
clinical diagnosis, 60, 160
clinical examination, 123
clinical presentation, 142
clinical symptoms, 83
closure, 73, 75, 76, 77, 85
CNS, 87, 90, 140
CO_2, 24, 42, 43, 92, 97
coagulopathy, 2, 19, 132
coding, 52
collateral, 2, 3, 4, 160
colonization, 84
coma, 140
compartment syndrome, 69, 71, 72
compliance, 85, 86, 113
complications, 5, 6, 14, 17, 39, 47, 97, 109, 117, 122, 127, 128, 130, 135, 169, 171
compressibility, 4, 18
compression, 20, 86, 90
computed tomography, 7, 83, 166
configuration, 75, 77
congestive heart failure (CHF), 12, 13, 91, 118, 168, 170
consciousness, 81
consent, 13, 155
constrictive pericarditis, 61
consumption, 7, 99, 168
containers, 155, 156
contamination, 102
cooling, 16, 143, 144, 145, 146
cor pulmonale, 61
coronary arteries, 41

cosmetic, 121, 122
cost effectiveness, 122
cough XE "cough" reflex, 90, 112
coughing, 13, 14, 156
CPP, 9, 81, 101
craniotomy, 8
cricoid cartilage, 24, 124
cricothyroid membrane, 24
Critical Care Medicine, ix, 15, 80, 105, 138
Critical Care Physicians, ix
Critical Care specialists, ix
critically ill patients, ix, 1, 15, 61, 72, 73, 94, 122, 128, 129, 130, 139, 141, 142
CSF, 83, 84, 86, 107, 108, 109
CT scan, 7, 8, 83
culture, 116
cure, 146
cyanosis, 95
cyst, 107
cytology, 11, 13

D

decerebrate rigidity, 146
deficiency, 43
deflate, 34, 35, 92, 132, 133
deflation, 73, 75, 76, 77, 78, 79, 133
dementia, 45
dentures, 91
depression, 93, 94, 140
depth, 19, 24, 29, 54, 55, 128, 133, 156
detection, 166
dialysis, 18, 139, 140, 141
diaphragm, 34, 41
diarrhea, 134
diastole, 73, 75, 77
diastolic blood pressure, 1
diastolic pressure, 75, 133, 135, 137
differential diagnosis, 120, 157
dilation, 34, 122, 123
discomfort, 1, 53, 93, 146, 156
displacement, 8, 21
dissociation, 102, 169
distress, 90, 153
distributive shock, 60
diuretic, 118
doppler, 161
drainage, 27, 28, 29, 31, 40, 41, 70, 83, 84, 101, 102, 155
dressings, 67
drugs, 1, 2
dyspnea, 27, 113

E

echocardiogram, 51, 52
edema, 90, 91, 100
effusion, 27, 29, 30, 52, 153, 155, 156
Egypt, 128
EKG, 19, 93, 140
electrolyte, 139
emboli, 64, 79, 91, 160
embolism, 6, 20, 67, 79
embolization, 64
emergency, 25, 37, 93, 98, 123, 137, 152, 164, 166, 170
emergency physician, 166
emphysema, 31
empyema, 27, 28, 30, 155, 156
encephalopathy, 140, 143, 144
endocardium, 149
endoscope, 47
endoscopy, 46
energy, 73
epidermis, 156
epiglottis, 90, 96
epinephrine, 2, 24, 64, 106, 124, 125
equilibrium, 81
equipment, 2, 23, 24, 64, 92, 123, 124, 155
esophageal varices, 37
esophagitis, 48
esophagus, 34, 35
etiology, 115, 153
evidence, 3, 57, 58, 60, 61, 79, 117, 163
examinations, 160
extensor, 5
extensor digitorum, 5
extraction, 102, 168, 169
extravasation, 65, 67
exudate, 120, 156, 157

F

fascia, 124
fat, 13, 64
fatty acids, 146
fauces, 90
femur, 63, 64
fever, 115
fiber, 11, 14, 97
fibrin, 102
fires, 150
first generation, 46
fixed rate, 150
flail chest, 90

flex, 18, 66
fluctuations, 71
fluid, 8, 11, 13, 27, 28, 29, 30, 31, 41, 61, 67, 70, 81, 83, 107, 115, 116, 117, 118, 119, 132, 141, 154, 155, 156, 157, 166, 169
fluid balance, 70
fluid management, 132
food, 91
food intake, 91
football, 34
formation, 43, 79, 102, 107, 135
fremitus, 155
fresh frozen plasma, 43
fungus, 13

G

gastric lavage, 46
gastric outlet obstruction, 46
gastrojejunostomy, 45
general anesthesia, 90
Georgia, 9, 147
glottis, 90, 93, 96
glucose, 156
goiter, 122
grading, 71
gravitational effect, 40
gravity, 39
gray matter, 8
growth, 66
guidance, 4, 17, 18, 20, 107, 116, 117, 124, 128, 130, 131, 132, 155
guidelines, 32, 49, 143, 157, 170

H

HCC, 117
head injuries, 9, 93, 94
head injury, 9, 87, 90
head trauma, 100
headache, 81, 109
health, 109
heart block, 152
heart rate, 124, 146, 150, 151
height, 53, 70, 78, 92
helium, 74
hematemesis, 90, 91
hematocrit, 100
hematoma, 8, 86, 90, 117, 135
hemodialysis, 15, 140, 141
hemodynamic instability, 76, 111
hemoglobin, 12, 42, 99, 102, 167, 168, 169
hemoptysis, 12, 90
hemorrhage, 4, 13, 23, 37, 42, 83, 100, 121, 123, 134, 144
hemothorax, 27, 28, 30, 31, 156
hepatic encephalopathy, 115, 143
hepatitis, 118
hepatorenal syndrome, 116, 117
hernia, 34
herpes, 154
herpes zoster, 154
heterogeneity, 140
histamine, 94
hub, 5, 20, 156
hydrocephalus, 83
hyoid, 24, 89
hyperemia, 100, 102
hyperglycemia, 151
hyperkalemia, 94, 140
hypertension, 69, 71, 72, 81, 83, 86, 100, 141
hyperthermia, 94, 144
hyponatremia, 117
hypotension, 13, 70, 81, 114, 117, 144, 166
hypotensive, 51, 52, 91, 93, 163, 164
hypothermia, 99, 143, 144, 146, 147, 169
hypovolemia, 60, 117, 119, 134
hypoxemia, 12, 14, 23, 41
hypoxia, 9, 70, 91, 95, 151, 167

I

ice packs, 144
ICS, 53, 55, 56
iliac crest, 63, 106
image, 53, 54, 55, 56, 59, 120
images, 11, 52, 53, 54
incidence, 17, 19, 127
incisors, 89, 97
India, 103
induction, 91, 93, 95, 143, 147
infants, 19, 63
infarction, 47, 99, 118, 135, 137, 161
infection, 5, 17, 19, 25, 64, 83, 84, 87, 95, 101, 106, 115, 116, 117, 118, 122
inferior vena cava, 52, 55, 59, 61, 166
inflammation, 108, 153, 169
inflation, 34, 47, 75, 76, 77, 79, 135, 136, 137
informed consent, 46, 116
inguinal, 4, 19
initiation, 139, 140, 142
injury(ies), 1, 7, 9, 20, 24, 25, 28, 30, 41, 43, 53, 66, 67, 81, 90, 96, 97, 99, 100, 101, 102, 121, 122, 127, 139, 142, 143, 144, 147, 156, 169
innominate, 122, 128

insertion, 8, 19, 20, 29, 31, 32, 47, 64, 67, 101, 106, 108, 116, 128, 132, 137, 145, 155, 156, 157
institutions, 143
Intensive Care Specialist, ix
intensive care unit, 7, 8, 9, 11, 111, 121, 122, 129, 130, 131, 159, 170
Intensivists, ix, 90
interface, 113
interference, 52
interphase, 112
intervention, 51, 74
intoxication, 108, 140
intra-aortic balloon pump, 73, 80
intracranial pressure, 83, 100, 105, 144, 146
intravenously, 63
iodine, 24, 116, 155
ipsilateral, 18, 30, 116
irradiation, 122
ischemia, 2, 7, 12, 70, 79, 102, 161, 162

K

kidney, 41, 139, 142
knees, 106

L

laceration, 117, 130
lactate level, 167
laryngoscope, 92, 96, 97
laryngoscopy, 34, 90, 92, 96
larynx, 24, 97
lateral epicondyle, 66
lead, 122, 150, 152
leakage, 107
leaks, 29, 34, 112, 113, 132
left atrium, 56, 58, 59
left ventricle, 56, 57, 58, 59, 60
legs, 107
lesions, 13, 69, 86, 161
ligament, 4, 19, 24
light, 11, 92, 153
liver, 42, 118, 140, 141
liver disease, 42, 118
liver failure, 141
local anesthesia, 19, 64, 106, 125
local anesthetic, 17, 28
low risk, 41
lumbar puncture, 109
lumen, 3, 15, 16, 17, 20, 40, 44, 74, 132, 159
lung function, 27, 41
lymphoma, 117

M

macrophages, 13
magnetic resonance imaging (MRI), 130, 161
malignancy, 29, 42, 45, 117, 118, 156
management, 1, 23, 32, 37, 41, 51, 52, 61, 71, 87, 91, 121, 131, 132, 140, 143, 147, 150, 170
mandible, 89, 96
marrow, 63, 65, 67
Marx, 120
mass, 83
mastoid, 101
mean arterial pressure, 69, 81, 100, 135
measurement, 1, 70, 71, 83, 102, 166, 169
measurements, 1, 71, 107, 136, 161
mechanical ventilation, ix, 14, 29, 39, 42, 114, 121, 122, 129, 130, 144
media, 47
medical, ix, 25, 32, 72, 109, 137, 150
medication, 45, 63
medicine, ix, 6, 22, 25, 32, 120, 152, 157, 164
meninges, 102
meningitis, 109
meta-analysis, 114, 119, 120, 122, 142, 164
Metabolic, 105
metabolic acidosis, 140
metabolism, 99, 102, 169
methemoglobinemia, 169
methodology, 140
Mexico, 105
microdialysis, 9
migration, 137, 151
misuse, 37
mitral insufficiency, 74
mitral valve, 56, 57
mixing, 42, 102
morbidity, 45, 87, 119, 139
mortality, 45, 139, 140, 143, 168, 169
multiple factors, 69
muscle relaxant, 93
muscles, 30, 56, 93
musculoskeletal, 53
myasthenia gravis, 90
myocardial infarction, 73, 80, 150, 168, 170
myocardial ischemia, 151
myoclonus, 146

N

nares, 90
nasogastric tube, 35
nausea, 81

neck injury, 90
necrosis, 35, 114
nerve, 4, 24, 156
neurological disease, 42
New Zealand, 51
North America, 32, 157
nutrition, 15, 16, 45, 49

O

obesity, 49, 89, 130
obstruction, 20, 23, 46, 48, 49, 67, 75, 90, 108, 118, 121, 136, 153
occlusion, 2, 5, 118, 133, 135
omentum, 118
opiates, 17, 93
orbit, 101
organ, 69, 71, 140, 142, 169
organs, 41, 69
osteogenesis, 64
oxygen, 7, 8, 9, 12, 15, 16, 19, 24, 39, 41, 42, 43, 73, 76, 77, 78, 79, 95, 99, 102, 103, 123, 124, 167, 168, 169, 170, 171
oxygen consumption, 8, 39, 79, 99, 167, 168
oxyhemoglobin, 40, 99

P

pacing, 149, 150, 151, 152
pain, 93, 115, 117, 146, 157
palate, 90, 96
pallor, 4
palpation, 4
pancreatitis, 72, 118
papilledema, 81
paracentesis, 116, 117, 118, 119, 120
paradigm shift, 131
parenchyma, 7, 8, 31
partial thromboplastin time, 74
pathology, 12, 83, 107, 153
pathways, 160
PCP, 13
pelvis, 4
perforation, 25, 47, 48, 118, 152
perfusion, 9, 41, 69, 71, 73, 76, 77, 78, 79, 81, 99, 101
pericardial effusion, 52, 55, 56, 57, 58, 60
pericardiocentesis, 52
pericarditis, 140
pericardium, 57
perinatal, 144
periosteum, 30
peripheral vascular disease, 74
peritoneal carcinomatosis, 118
peritoneum, 4, 117
peritonitis, 115, 117
pH, 42, 140, 156
pharynx, 96
Philadelphia, 7, 49, 63, 81, 89, 99, 120, 137, 138, 152, 159, 161
physical therapy, 122
physicians, xi, 52, 89, 164
physiological, 151
physiology, 52, 60, 81
pilot study, 120, 147
placebo, 114
plaque, 74
plasmapheresis, 16
platelets, 116, 123
pleura, 29, 30, 32, 156, 157
pleural cavity, 156
pleural effusion, 27, 28, 29, 32, 54, 55, 57, 58, 60, 153, 156, 157
pleurodesis, 155
pneumonia, 35, 71, 129
pneumothorax, 18, 19, 27, 28, 29, 30, 31, 92, 127
polyurethane, 2, 47
population, 52, 72, 130
positive end expiratory pressure, 123
pregnancy, 118
prevention, 21, 129
probe, 8, 17, 53, 54, 123, 145
prognosis, 72, 116, 140
prophylactic, 28
prophylaxis, 21, 46
pulmonary artery pressure, 136
pulmonary capillary wedge pressure, 133, 135
pulmonary edema, 41, 114, 131, 140, 156
pulmonary embolism, 43, 60
pulmonary hypertension, 42, 132
pumps, 42

Q

QRS complex, 151
quadriceps, 66
quantification, 61

R

radiation, 34
radius, 63
rape, 28
reactions, 94

real-time basis, 160
receptors, 140
recognition, 1, 143
recovery, 74, 140, 141, 143, 144
recruiting, 111
rectal temperature, 145
reflexes, 94
regionalization, 146
relaxation, 75, 96, 134
renal failure, 117, 132, 139
renal replacement therapy, 16, 139, 141, 142
renin, 117
requirements, 42, 79, 123
resection, 49
resistance, 18, 34, 40, 43, 65, 99, 101, 107, 112, 113, 135, 159
resolution, 53
resource availability, 141
respiratory failure, 39, 41, 42, 43, 44, 114
respiratory rate, 112, 113, 124
respiratory therapist, 92
response, 61, 113, 146, 163
responsiveness, 61, 118, 163, 166
RH, 43, 44, 130
rheumatoid arthritis, 97
rhino, 129
rhythm, 150
right atrium, 40, 41, 58, 59, 133, 151
right ventricle, 56, 57, 58, 59, 133, 135, 149, 151
rings, 24, 121, 124, 125
risk, 6, 12, 18, 19, 24, 28, 34, 53, 64, 66, 69, 71, 74, 75, 79, 82, 84, 91, 93, 100, 117, 122, 123, 127, 128, 130, 152, 154, 156, 160, 161, 170
risk factors, 6, 69, 130
risks, ix, 24, 34, 87, 128, 155, 168
RVS, 135

S

safety, 14, 117, 121, 128, 130, 156
saturation, 9, 12, 15, 16, 39, 41, 42, 95, 99, 102, 103, 113, 124, 167, 168, 170, 171
sclerosis, 37
scoliosis, 122
scope, ix, 11, 13, 52, 59, 153
secretion, 122, 137
sedative, 46
sedatives, 93, 96
seizure, 93
sensation, 65
sensing, 151
sensitivity, 8, 118, 150, 151, 160, 161, 164
sepsis, 60, 61, 74, 134, 135, 146, 167, 169, 170, 171
septic shock, 60, 61, 71, 167, 169, 170, 171
septum, 60
serum, 118, 120, 156, 157
shock, 60, 131, 134, 135, 137, 164, 166, 167, 169
showing, 75, 82, 107, 159
sickle cell, 161, 162
signals, 160
signs, 52, 69, 81, 89, 114, 140
silk, 92
silver, 13
simulation, 21
skin, 2, 5, 8, 17, 18, 19, 24, 30, 47, 65, 107, 116, 117, 124, 125, 126, 128, 154, 156
sodium, 65, 70, 140
solution, 17, 65, 106, 155
sphygmomanometer, 34
spinal cord, 143, 147
spinal cord injury, 143, 147
spine, 4, 24, 91, 95, 96, 97, 100, 116, 122
spleen, 117
sputum, 13
stability, 141
states, 60,131, 134, 167, 168
status asthmaticus, 43
status epilepticus, 90
stenosis, 24, 97, 121, 127, 160, 161
stent, 37, 74
sterile, 17, 18, 27, 28, 29, 30, 47, 106, 124, 132, 133, 155, 156
sternocleidomastoid, 18, 101
sternum, 55, 63, 64
stethoscope, 47
stomach, 34, 47, 117
stress, 77, 93
stretching, 20
stroke, 45, 77, 78, 90, 122, 144, 147, 159, 160, 161, 162
stroke volume, 77, 78
structure, 24, 54
subarachnoid hemorrhage, 7, 9, 105, 107
subcutaneous emphysema, 25, 127
subcutaneous tissue, 5, 65
subdural hematoma, 107
superior vena cava, 18, 133, 167, 171
supervision, 83
support staff, 123
suppression, 93, 144
surface area, 42
surgical technique, 122
survival, 71, 119, 129, 141, 147
survivors, 147
suture, 2, 5, 20, 29, 30, 47
Swan-Ganz catheter, 131

swelling, 65
symptoms, 81, 140
syndrome, 2, 6, 67, 79, 93, 118, 142
systolic blood pressure, 1, 73, 83
systolic pressure, 75, 78, 133, 135, 137

T

target, 13, 17, 142
TBI, 7, 83, 100, 102
teachers, ix
techniques, 61, 121, 122, 128, 129
teeth, 91, 96, 97
temperature, 143, 144, 145, 147
tendon, 5
tension, 7, 9, 12, 28, 34, 113
therapeutic interventions, 144
therapy, 61, 114, 116, 140, 141, 142, 166, 169, 170, 171
thoracentesis, 32, 153, 154, 155, 156, 157
thoracoscopy, 32
thoracostomy, 27, 28
thrombocytopenia, 28, 154
thrombosis, 21, 102, 166
thrombus, 79, 135, 137
thyroid, 18, 24, 89, 96, 127
tibia, 63, 64, 65
tissue, 7, 8, 9, 18, 29, 30, 39, 53, 65, 82, 102, 106, 122, 147, 156, 167, 168, 169
tissue perfusion, 39
trachea, 11, 13, 24, 97, 121, 122, 126, 127, 128
tracheostomy, 11, 24, 111, 121, 122, 124, 126, 127, 128, 129, 130
transcranial Doppler sonography, 161
transducer, 3, 5, 19, 53, 55, 56, 57, 59, 70, 83, 84, 136, 137
transection, 23
transport, 170
trauma, 23, 45, 48, 69, 96, 122, 130, 144, 170, 171
traumatic brain injury, 7, 9, 83, 100, 102, 143, 146
treatment, 14, 27, 37, 52, 61, 72, 103, 109, 142, 171
trial, 37, 39, 41, 43, 44, 114, 128, 129, 130, 142, 146, 147, 170, 171
triglycerides, 156
tumor, 23, 49, 86, 90, 108, 122

U

ultrasonography, 128, 161, 162, 166
ultrasound, 4, 17, 18, 19, 20, 28, 51, 60, 61, 100, 107, 116, 117, 121, 123, 128, 130, 132, 155, 159, 161, 163, 164, 166
unstable angina, 74
unstable patients, 1, 4, 122, 141
urea, 140
urinary bladder, 70
urinary tract, 71
urinary tract infection, 71
urine, 70, 79, 140
uvula, 90

V

valve, 24, 92, 132
vapor, 43
vasodilation, 135
vasopressor, 16
vasospasm, 100, 103, 159, 161
vein, 4, 9, 15, 17, 18, 20, 21, 41, 59, 65, 74, 101, 102, 132, 133, 141, 145, 149, 163, 164, 166
velocity, 159, 160, 161
ventilation, 39, 43, 45, 89, 91, 95, 98, 111, 112, 114, 122, 127
ventricle, 59, 77, 83, 135, 149, 151
ventricular septal defect, 74
vessels, 4, 81, 127, 128, 159, 160
viscera, 69
viscosity, 99
vision, 96
visualization, 4, 34, 59, 95, 121, 125, 126, 127
vomiting, 81, 134

W

water, 29, 31, 43, 47, 132
weakness, 90
white matter, 8
windows, 159, 161
working hours, 123

X

xiphoid process, 146